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CONTENTS.

Editorial 279
Editorial Comment 282
Original Articles 283
Some Heart Problems Suggesting the Necessity for a Closer Alliance Between the Physiologists, Bio-Chemists and Clinician. By Wm. Watt Kerr, M. D..... 283
Syphilitic Arthritis. By L. W. Ely, M. D..... 288
The Tonsils as a Focus on Infection. By J. McK. Brown, M. D. 290
Focal Infection—Intestinal Involvement. By F. F. Gundrum, M. D..... 293
High Calory Feeding in Cases of Typhoid Fever in Children. By H. H. Yerington, M. D. 294
Clinical Observations of One Hundred Cases of Artificial Pneumothorax. By R. C. Matson, M. D..... 297
Tuberculosis in Childhood with Unusual Manifestations. By Langley Porter, M. D..... 303
Differential Diagnosis of Abdominal Tuberculosis. By George E. Ebright, M. D.... 306

(Contents continued on page IX.)

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THE RED CROSS.

"Decisive victories have not yet followed the flags of the central nor the allied armies, but in all, the Red Cross signalizes the most triumphant achievement of man. International laws have been torn into shreds and become mere scraps of paper, moral and religious precepts and codes have been supplanted by brutalities never practiced by primitive man, and the foundations of civilization have seemed to be on the point of disruption and final collapse, but the spirit and ideals of scientific medicine remain unsullied, and a new world in which these shall dominate, will be created." In these words Dr. Victor C. Vaughan (Graves, So. Med. Jour., 1917, X, 4) marks the present position of the Red Cross and thereby the duty which every physician owes to it.

Duty is the highest word in the physician's vocabulary. In the war which is upon us, duty demands certain definite things from every physician. Too many do not appreciate the seriousness of the times. Too many do not realize that if this war is not fought to a successful finish in Europe it will with absolute certainty be finished on American soil. Our profession has responded well to the insistent demands from home and abroad. No word of censure is intended for past performance. It is most creditable. But the future is what concerns us and not what is past. Every physician under thirty years of age or who has graduated within the last four years should have a most excellent excuse if he is not already enrolled in one of the services. It is to be hoped that no physician will be actually drafted for the new army. The medical profession is a volunteer profession. Let us live up to our standard.

So much for those who can go. A larger proportion cannot go, either because of age and health disabilities, or because of valid personal or public reasons. These have an opportunity which none should miss of enrollment with the Red Cross. Full information can be secured from the officers of the various county medical societies as to local Red Cross agencies. Suffice it here to say that the national Red Cross is under governmental supervision. The President is its head. Its accounts are audited by the War Department. Its extension and activity depend on subscriptions from members. Every community in the state should have an active Red Cross branch. Here is one opportunity for the physician, especially in suburban districts. It is in a strong sense his responsibility to see to this. Let him get in touch through his county society with Red Cross headquarters at once, and see that his community is organized. Branches are organized according to geographic location alone and each branch carries on all the varieties of Red Cross activity which are practicable or needed in its territory. Not only does each branch charge itself with increasing its membership and contributing to war and relief needs of the nation, but it is a permanent organization with authority for relief and promise of assistance and co-operation in case of any local calamity or disaster.

Under the regulations governing the American Red Cross in time of war, the various Red Cross units serving with the United States land forces become an integral part of the sanitary service of the land forces, and their members become subject to military law and discipline, and are entitled to the rights and benefits of their appropriate rank in the sanitary service. Except in great emergency, Red Cross units will not be ordered to the front but will be assigned to the zone of communications, base hospitals, home country hospitals and hospital ships. At the call of the War Department, the Red Cross furnishes organized units, sections, detachments and individuals, whose service may be required. These may be physicians, nurses, surgeons, dentists, chaplains, stenographers, pharmacists, laboratory technicians, clerks and hospital personnel.

Whenever lawful authority permits, persons absorbed by the army sanitary service from the Red Cross will be paid according to the rank held. Others known as Red Cross volunteers serve without pay but are provided with subsistence, transportation, and quarters. All are subject to army discipline and law. The Red Cross personnel is enrolled in three classes,—Class A, those willing to serve wherever needed; Class B, those willing to serve in home country only; Class C, those willing to serve at place of residence only. Those enrolled in Red Cross units designed for service in base hospitals or in the zone of communications must be in Class A.

Eleven units are organized by the Red Cross for service with the army or for training personnel. These are ambulance companies, base hospitals, hospital units, surgical sections, emer-

gency nurse detachments, sanitary training sections, information sections, refreshment units and detachments, supply depots, general hospitals, and convalescent homes. Details of these various organizations will be given later. Volunteers, both men and women, have a long list of activities from which to select their preference, for service anywhere, in the United States only, or at residence only. The work of the Red Cross is so varied that every person can find something he can do, no matter what his circumstances, and can do it with the certain knowledge that it will count. Physicians can organize, teach in Red Cross instructional courses, help administer, assist in publicity work, or engage in other available local activities, even if they can not enroll for service with the sanitary forces.

THE ALCOHOL QUESTION.

II. ALCOHOL AND THE WAR.

Several ways present themselves whereby the alcohol question assumes importance in war. Moreover these considerations have a peculiar significance in their immediate application to war conditions in the United States today. What is the direct influence of alcohol on the individual efficiency of the fighting forces? That question must be squarely answered. What are the indirect results of the use of alcohol, for instance as regards increased susceptibility to disease, and increased exposure to prostitution and venereal disease? Finally is it logical to preach food conservation and allow the present quantity of foodstuffs to be used for manufacture of alcoholic beverages? In short, is alcohol going to help materially in winning the war, or can it have any influence on the war? The former questions were in part answered editorially last month. The last question especially concerns us here.

Numerous estimations have appeared regarding the bulk of food materials used for manufacture of alcoholic drinks. A conservative and authoritative statement seems to be furnished by the War Prohibition Committee, consisting of Professors Carver, Day, Ripley and Gay of the Harvard University Department of Economics, and Dr. Irving Fisher, professor of political economy in Yale University. (Am. J. Pub. Health, 1917, June, VII, 581). This statement quotes from the report of the Commissioner of Internal Revenue for the fiscal year of 1916 (pg. 138) a total of 3,603,911,916 pounds of molasses and grain used in that year for the production of distilled liquors. This excludes wheat, barley, oats, and "other materials" aggregating 82,150 pounds. On the basis of carefully studied data, the committee estimates that for the same year 3,390,399,219 pounds of the same food materials were used for the manufacture of fermented liquors. Thus it appears that in the fiscal year of 1916 a total of 6,994,311,135 pounds of grain and molasses were used in the manufacture of fermented and distilled liquors in the United States, not to mention fruits, glucose and minor ingredients.

These figures were reviewed by a second committee composed of Professors Percy G. Stiles, Walter B. Cannon and Irving Fisher, who state that probably one-sixth of the total of seven billion pounds would be necessary for the production of denatured alcohol, and that the remaining five-sixths by conservative estimate would supply the caloric requirement for a year of seven million men.

In a letter to the New York Times (May 27, 1917), Professor Fisher emphasizes that the manufacture of these grains into alcoholic beverages results in a loss of approximately eleven million one pound loaves of bread each day, enough to furnish one pound of bread per man daily to the combined armies of England and France. He states further that this loss represents "5½ per cent. of the total food values consumed by human beings in the United States, over 10 per cent. of the total consumption of breadstuffs in the United States, 13 per cent. of the wheat consumed in the United States, and 25 per cent. of the wheat exported from the United States in 1916."

These figures are not to be held lightly. If there is occasion in fact,—and adequate surveys seem to indicate that there is,—for national control of production and distribution of food supplies to the end of averting definite food shortage for ourselves and our allies, then *pari passu* the question of manufacture of alcoholic beverages becomes of vital moment. How far such manufacture should be restricted and for what period it should be restricted can only be decided rightly by expert judgment of all the factors concerned, such as the nature and degree of food shortage, and the temporary dislocation of industry and paralysis of capital which might follow. Parenthetically it may be said that this dislocation of industry could in no wise approach that resulting from calling to the colors even the first contingent of the new army. Moreover the removal of the army drafts from industry would more than supply occupation to the group thrown out of employment by cessation of alcoholic manufactures. Nor in a generation of "war brides," war shipping opportunities and all the unusual demands added to the ordinary opportunities for capital investment, is a serious paralysis of capital to be feared.

It is a matter of pertinent interest that at its annual meeting in Cincinnati, May 9-11, 1917, the National Association for the Study and Prevention of Tuberculosis adopted the following resolution, "That this Association place itself on record as favoring national prohibition both for soldiers and civilians during the war period and for one year thereafter." With a distinguished membership and attendance drawn from the most eminent of America's physicians, the American Society for Clinical Investigation unanimously adopted the following resolution at its annual meeting at Atlantic City in May, 1917: "Resolved, that in the critical condition of the world's food supply we consider it desirable that the manufacture of alcoholic beverages and their importation into this

country be prohibited for the duration of the war and for at least one year thereafter." These resolutions are examples of many and illustrate the present trend of medical and scientific opinion based on the present conception of the action of alcohol, and of the relation of alcohol to the conduct of war.

THE MEDICAL DEFENSE RULES.

The Secretary's office has mailed to each member of the Society a printed copy of the MEDICAL DEFENSE RULES. Members have been requested to familiarize themselves with the Rules, and we wish to impress again upon the members how much it is to their interest to do so.

The compilation and restatement of our MEDICAL DEFENSE RULES and putting them in the hands of every member is one of the most important steps taken by the Council for the welfare of the entire Society. While the general tenor of some of the Rules has been known to nearly all of our members, the exact text has never been carefully and accurately stated and placed directly in the hands of each member. Heretofore, discussion has been had in sporadic cases as to the application of a given rule, which would have been avoided had members been given an opportunity to familiarize themselves with these provisions. It is earnestly suggested to our members that strict compliance with the rules will do much for the benefit of the Society; and in a particular case it may be vital to the individual's interest.

We have already taken occasion to comment in these columns upon the rule regarding election between defense by an insurance company (if a member is insured), and defense by the Society. We again repeat that this rule was adopted and has been kept in force solely because the funds are not available to afford defense by the Society in every case. Therefore, the Council felt that in requiring members to make such election the greatest good was given to the greatest number. We trust, that as our organization grows, it will be possible to revoke this rule.

We have also, from time to time, heard and participated in discussions concerning the so-called X-ray rule. Members seem to be more or less apprehensive that this rule will deprive them of defense by the Society in cases arising out of treatment of injury to bone or joint. The rule has no such effect. It expressly provides that the discretion of the Council should be exercised in every case where any reasonable grounds exist for excuse of failure to take X-ray plates. The Council uniformly exercises a sane and common sense judgment and excuses such omission. Of course, every case must depend upon itself; and a member cannot expect such favorable ruling if he recklessly and intentionally failed to exercise reasonable and ordinary precaution.

May we ask the members in considering the Rules to remember that they represent the combined judgment of men, most of whom have been at the head of the Society's affairs for many years, and who have had the benefit of all the precedence available, to-wit: eight years of experience throughout the life of our Legal Department.

MEDICAL WOMEN AND THE WAR.

Medical women have organized in various parts of the country for the purpose of offering their services to their country in the present emergency. Unfortunately no means has as yet been devised whereby the government or any of its allied bodies can take advantage of these offers, because there is no recognition of physicians of the female sex in either the Army or the Navy. This, of course, prevents the Red Cross from using these women to the full in capacities where they might excel because they are unable to join the Medical Reserve Corps of the Army or Navy. This is exactly as it was in England at the outbreak of the war three years ago. The English medical women, in spite of the inability immediately to get recognition, were able, because of the pressing need, to establish hospitals in France and so to demonstrate their ability to be of real and valuable service to the cause for which their men were giving their lives. After that they were enrolled definitely as a part of the medical staff of the army and were given the rank to which their positions entitled them.

We know of one woman (not a physician) who served in France as a Nurse's Aid with the rank of Corporal, and of another who was Matron in a base-hospital with the rank of Colonel in the British Army. We understand that the pay of these women is not quite that of men of the same rank, but cannot state this authoritatively.

We know that in this country women physicians have been asked by representatives of the Red Cross to organize groups to aid, without pay or rank, in the rehabilitation work in France. This we feel to be inconsistent. If the Government, or the Red Cross, does not wish the services of women for any particular reason, well and good, but if these services are to be used, there should be no distinction. *She* is just as much a surgeon when in the operating-room as is *he*. Why should John Smith, M. D., an anesthetist, be a Second Lieutenant, and Mary Brown, M. D., an anesthetist, be a Nurse's Aid without authority?

REGISTRATION FEES.

Every licentiate has received a slip announcing that a registration fee of two dollars per annum has been imposed upon them by the legislature at the recent session. While this is presumably a "registration fee," it is really a tax for raising funds for the prosecution of violators of the Medical Practice act. The Board of Medical Examiners have not been able, with the funds accruing from the fines of convicted miscreants, to pursue a sufficiently vigorous campaign of elimination of

quacks and others operating contrary to the law.

While the Medical Practice act is designed primarily for the protection of the public, and while any taxation for this protection should be in the nature of a public tax, we find on inquiry that it is a sound principle of jurisprudence to tax a craft whose calling requires regulation in order to carry out such regulation.

The accompanying letter from the Secretary-Treasurer of the Board of Medical Examiners will explain the machinery by which the tax is to be collected.

BOARD OF MEDICAL EXAMINERS OF THE STATE OF CALIFORNIA.

Sacramento, California, July 3, 1917.

Dear Doctor:

Replying to your recent inquiry beg to advise that among the amendments passed by the 1917 Legislature was one providing for a $2.00 registration fee payable to the Board of Medical Examiners by all holders of any form of certificate issued by this or prior boards regulating the healing art in California.

The first payment of this fee is due January 1, 1918, and subsequently on January first of each year. Failure to pay the fee within 60 days after January first of each year automatically revokes the certificate and a fee of $10.00 must be paid to the Board of Medical Examiners in order to restore such certificate as may be thus revoked.

Thirty days prior to January first of each year the Board expects to forward a notice to all certificate holders, and in order to insure delivery of such notice, the licentiates must keep the office of the Board of Medical Examiners notified of each change of address, lest the notice fail to be delivered and the payment of the fee be neglected.

The Board expects to forward a copy of the directory to be published January first of each year, to each individual licentiate who forwards his fee as above noted.

Very truly yours,

(Signed:)

CHARLES B. PINKHAM, M. D.,
Secretary-Treasurer.

EDITORIAL COMMENT.

When motoring, camping or picnicking, the first desideratum for a safe and sane outing is a safe and sane water supply. What doth it profit a man to gain the whole wide out-doors and contract a typhoid or dysentery infection? Summer typhoid is rightly named. Autumnal fever was rightly named. And the reason is well-known, when we stop to consider. If a physician is only a physician in his consulting room and professional rounds, then better for all were he not a physician at any time. He it is who must see that the picnic has safe water to drink and that the outing party does not risk its individual health on sewage which it mistakes for a beverage.

In the June bulletin of the California State Board of Health, Frank Bachmann reiterates that sparkling clear water is more apt to be a disease carrier than grossly contaminated water. All surface waters are more or less subject to dangerous infection. He states that typhoid bacilli may live thirty days in water of ordinary temperature and much longer in colder waters. Bachmann emphasizes the advantage of boiled water as the safest when boiling is practicable. When boiling is not feasible, he recommends the use of tincture of iodine, three drops to the quart of clear waters, as an efficient bactericide. If the water is cloudy or contains much sediment, six drops should be used per quart, or enough to produce a very faint brownish discoloration. After the iodine has acted for fifteen minutes, a pinch of ordinary photographic "hypo," (sodium thio-sulphate), clears the water and removes the last traces of iodine. The fifteen minute interval is important and also the brown color. In this strength there is no disadvantage from the chemicals used.

If typhoid is a sanitary crime, then is the physician an arch-criminal if he does not insure safe drinking water for outing parties with which he may be associated. With the present record of typhoid vaccination, every physician should be an example of such vaccination and should preach it to his friends and patients. Especially should he do this, since the State Board of Health will furnish the vaccine free of charge. Why not use the Board of Health more and become missionaries of the prevention of disease by such means as have here been enumerated?

As man pursues his onward and upward march, so does he tend to limit the hirsute endowment of his face, even as nature tends to limit the hairy protection of his cranium. A full beard is rare enough to excite comment and as a sanitary measure in modern camp and war conditions, the beard has had its day. Even the mustache is becoming scarce and has to answer the same indictment as its full-blown progenitor. Beards are said to serve but three functions—to heighten confidence in the masculinity of the owner, to cover a feeble chin, or to incite confidence in the beholder as to the age of the bearded. Perhaps all three motives are present to a mixed degree. Perhaps, too, there is an element of disinclination to exertion necessary to be clean shaven, or even of desire for a difference from the common fashion, which last may be after all a tenable argument. In any case, the institution of the beard seems doomed under modern battle conditions and does not bear the scrutiny of the microscope or the aseptic conscience. Down with the beard and may we as a profession be clean shaven, clean principled, and unashamed either of our chins or of our practiced ethics.

The National Committee for Mental Hygiene has created a subcommittee on furnishing hospital units for nervous and mental disorders to the United States Government, the project having been approved by Surgeon General W. C. Gorgas of the U. S. Army.

This subcommittee, of which Dr. Pearce Bailey of New York is chairman, is authorized to secure the services of alienists and neurologists to be commissioned in the Officers' Reserve Corps, Medical Section, and to serve in the neuro-psychiatric units which are to be attached to the base and other hospitals of the military services of the United States. Further information will be given, and application forms sent to physicians qualified in this branch of medicine, on application to The National Committee for Mental Hygiene, 50 Union Square, New York City.

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The next evil which should be attacked with the utmost vigor by all boards of health is alcoholism. Public opinion needs to be enlightened on two points with regard to the use of alcohol as a beverage.

In the first place, it should be brought home to the entire population that the habitual use of alcoholic beverages reduces, in a serious degree, the productive efficiency of the community.

In the second place, recent experiments on the effects of alcohol on the nerves and glands of the human body have demonstrated beyond a doubt that alcohol invariably does harm, and never any good either in health or disease. The use of alcohol as a defense against exposure or fatigue has been given up by all sensible persons.

The evil is rooted, first, in what are called vested interests—that is, in the investment of large amounts of capital in the plants which produce, store and distribute beers, wines and spirits; and secondly, in the methods of taxation to which the white nations are accustomed. Heretofore the medical profession and the public health officers have given an uncertain sound concerning the use of alcohol.

It remains for the boards of health to attack this hideous evil with the weapons and in the spirit of preventive medicine. They should bring to the work all recent knowledge concerning the effects of alcohol on the human body, call to their aid legislators who can find equivalents for the public revenue now derived from the manufacture and sale of alcoholic drinks, and re-enforce to the utmost the wise counsellors who by moral teachings have brought about during the past fifty years considerable improvements in regard to the use of alcohol in the more intelligent and conscientious classes.—Haven Emerson, M. D., Amer. Jour. Pub. Health, June, 1917.

Original Articles

SOME HEART PROBLEMS SUGGESTING THE NECESSITY FOR A CLOSER ALLIANCE BETWEEN THE PHYSIOLOGISTS, BIO-CHEMISTS AND CLINICIAN.*

By WM. WATT KERR, M. D., San Francisco,
Professor of Clinical Medicine University of California.

For at least five years physiologists have agreed that lack of oxygen in the *blood* is rarely a stimulant to the respiratory center, but that the very slightest increase of carbon dioxide, resulting from any increase in the body processes, at once augments the rate and depth of respiration to such an extent that the additional inspired air furnishes enough oxygen to supply the new demand. Nevertheless, the great majority of clinicians still explain the symptoms of cardiac dyspnœa, and base their treatment on the older supposition.

In the *Journal of the American Medical Association* (November 4, 1911), the writer published a paper in which attention was called to the observations of Martin Flack, as well as those of Bachman, regarding the toxic effect of lactic acid upon the heart; likewise to the researches of Ryffel and others into the sources and fate of lactic acid in the human body, and expressed the hope that the laboratory work of these different investigators would lead to a more rational and successful cardio-therapy than that at present in vogue.

As full references to the work done by these gentlemen will be found in the article above mentioned it is unnecessary to repeat them in detail. Nine years ago Flack showed that if a frog's ventricle is placed in a weak solution of lactic acid (1 in 10,000 normal saline solution) the contractions become less and less, so that finally the ventricle stops in a state of complete relaxation; and about the same time Bachman, by perfusing rabbits' hearts with solutions of lactic acid obtained increased rate with greatly diminished force of contraction and a simultaneous dilatation of the coronary arteries, a fact that would indicate lactic acid to be a paralyzing agent for all muscles of the cardio-vascular system; neither did it require a strong solution, but even amounts so small as those occasionally found in the blood of a normal rabbit had some paralyzing effect.

One of the first thoughts suggested by these experiments is an interpretation of what we call acute dilatation of the heart as it occurs during exertion, a condition that is generally described as if it were a purely mechanical effect of "heart strain." Most of us are familiar with the breakdowns that take place during or immediately after an athletic contest, and have seen them in varying degrees of severity, some when the heart recovered its normal dimensions within two or three days, and others where the change was more prolonged or even permanent. Ryffel (*Quarterly Journal of Medicine*, Vol. 3, No. 10, January, 1910), found the lactic acid in urine from competitors in a

* Read before the St. Francis Hospital Clinical Society, March 31, 1916.

twenty-four hours' track walking race in no case above 6.5 mg. per hour, but obtained relatively large quantities from that passed following violent exercise, 430 mg., subsequent to 0.36 mile, and 818 mg. after 0.6 mile, running as fast as possible round a track thirty-three laps to the mile. The lactic acid in the blood, taken from a vein in the forearm immediately after the exercise, was also increased in one case from 12.5 mg. at rest to 70.8 mg. per 100 c.c. Excess of lactic acid disappeared from the urine in about thirty minutes after stopping, but in forty-five minutes the lactic acid of the blood was not quite reduced to normal. The older observers supposed that the production of lactic acid by muscle depended on its long-continued activity, but evidently this is not the case, and the important factor is the relation between the activity of the muscles and their supply of oxygen. The increased respiration of exercise has more effect in removing carbon dioxide than lactic acid, so that in course of time the carbon dioxide pressure becomes low, whilst the acidity of the blood is still high, and the latter is only gradually diminished after the cessation of the exercise by oxidation of the lactic acid and excretion of urine containing an excess of lactic acid, this process occupying about one hour after short periods of violent activity.

Here is some laboratory work that is absolutely pregnant with matters of importance to the clinician, and yet they are practically ignored by the vast majority. Take for example its relation to the hearts of athletes. It is exceptional in the universities and public schools that the hearts of members of the various athletic teams are examined after exercise; most of them are examined before admission to the team, and if the heart appears to be normal they are told to go ahead, and no further notice is taken of them unless they complain or show marked signs of distress. Each year, generally when the excitement of the season is over, for we all know how a boy hates to be a "quitter," a number apply for treatment on account of palpitation or some other cardiac discomfort, and it takes months to get their hearts reduced to the normal area, indeed some of them never do fully recover. It is highly improbable that these changes in the myocardium are due to a *simple* strain, such as might result from lifting a heavy weight, but rather they are the outcome of repeated intoxications of the cardiac muscle with lactic acid. It is not permissible to say that because acid is quickly eliminated during the period of rest that it therefore cannot do much harm; the effects persist after the elimination of the toxin; a fire travels quickly through a forest, but it takes a long time for the trees to grow again.

These observations on the production of lactic acid and its influence on the cardio-vascular system furnish us with a definite physiological basis on which we can frame an opinion regarding the nature and amount of exercise that may be allowed to different individuals. The term "heart strain" is so very vague that there is much difficulty in estimating what it constitutes, but since we know that exertion generates a toxin which is injurious to the myocardium, and as it has been demonstrated that the amount of toxin produced varies with the demand for *rapid* oxidation rather than with the duration of the effort we are in a much better position than formerly to decide what exercises are most trying to the heart. But the distinctive idiosyncrasy of each case must always be taken into account, for we all know that the susceptibility of normal hearts to toxins varies in different individuals, the influence of tobacco being a familiar example, and consequently much of this trouble could be avoided by examining the heart immediately after exercise. Because a boy cannot stand one form of athletic exercise it does not follow that he must be excluded from all others. Personal experience with participators in college sports has led me to the conclusion that track and oarsmen suffer most, then football and tennis players, and lastly, the baseball men; of course, it must be understood that these remarks apply to men who are exerting themselves under the stimulus of competition. At one time I thought it was otherwise, and used to forbid football where I allowed the more refined and gentle sport of track work, but experience made me change my opinion, and come to the conclusion that while the footballer took bigger chances on his bones and his neck, he was not so liable to injure his heart as was the sprinter and hurdler.

These observations on lactic acid also suggested the idea that much of the suffering experienced in senile hearts, associated with varying degrees of arterio-sclerosis, might result from the presence of lactic acid in the blood. The type of case is familiar to every physician. The patient is generally over fifty-five years of age, and in the early stage does not show any signs of dropsy or oedema, but his chief complaint is rather of weakness, or easily induced fatigue; he feels slightly dyspnœic on trivial exertions, such as bending over to lace his shoes in the morning, or performing his toilet, and the trouble may increase until not only is he always conscious that his breathing is abnormal, but paroxysms of increased severity are readily induced by excitement, exertion, or a variety of other causes. Or there may be gastric distress, such as a craving sensation in the stomach, but an absolute disgust for food the moment it is placed before him. The expression is keen and alert, altogether different from the dull condition so common in cases of valvular disease where there is pulmonary congestion with oedema, bronchitis and possibly pleural effusion as a result of failed compensation. Of course the two types may co-exist, but I believe an associated arterio-sclerosis is essential to the production of the first group of symptoms.

It may be of interest to the physiologist and biochemist to follow the vagaries of a clinician's mind in his endeavor to utilize their observations in the treatment of his patients.

The plan of campaign adopted, just prior to the writing of my paper in 1911, was two-fold: (1) To neutralize existing acids in the blood; (2) to find the source and fate of lactic acid in the body,

and try to influence those in the patient so that the balance between its formation and elimination might be restored.

It is now nearly five years since this treatment was begun, but I cannot remember any case where the patient's condition seemed to be improved by the use of alkalies; indeed the only two drugs that seemed to be of any permanent value were the iodide of potash and the protoiodide of mercury, both of which belong to the alterative group. The failure of soda to furnish relief was particularly disappointing because the good results so frequently attributed to its administration in the acidosis of diabetes has encouraged the expectation that not only would it aid in transporting carbon dioxide from the tissues to the lungs, but that, by increasing the alkalinity of the blood it would remove the irritation from the respiratory centre.

In his third Herter lecture, delivered at Johns Hopkins Hospital, October, 1914 ("Lectures on the Heart," by Thomas Lewis, published by Paul B. Hueber, New York), Dr. Thomas Lewis expresses the opinion that much of the cardiac dyspnœa is due to a diminished alkalinity of the blood from the presence of lactic and other acids at present unknown. He does not make any suggestions in regard to treatment, but further states that a characteristic of the alveolar air in these cases is "high oxygen and low carbon dioxide content"; that "the blood is fully aerated and has a low tension of carbon dioxide."

My conclusions from such findings would be that the symptoms cannot be attributed to lack of oxygen or excess of carbonic acid in the blood, but that there appears to be deficient oxidation in the tissues, either because the oxygen in the blood cannot reach them, or because, on account of the absence of some enzyme or other necessary agent, the tissues cannot utilize the oxygen supplied to them.

The presence of lactic acid in the blood is an evidence of deficient oxidation. The sources of lactic acid in the human body are the metabolism of glucose in muscles during their activity, and to a lesser extent from the proteid substance, and it also arises from glucose and other carbo-hydrates during constructive metabolism. That the destruction of lactic acid in the muscles and in the body generally depends upon an adequate supply of oxygen has been shown by several observers, and, as Stoklasa discovered a ferment both in muscle and blood that converted lactic acid into alcohol and carbon dioxide, it is possible that the oxidation takes place through some such indirect process. In the normal person an excess of lactic acid arising from severe exertion or other causes, stimulates the respiratory center until the greater ventilation of the lungs provides enough oxygen to compensate for this increase, and the subject therefore quickly recovers even from severe dyspnœa when he comes to rest. Furthermore, although the previous inhalation of oxygen enables an athlete to undertake exertion with much less discomfort, to the cardiac dyspnœic of the type under consideration the administration of oxygen brings no relief, and consequently we are forced to the belief that in his case some connecting link between the supply and utilization of oxygen has been lost. If it be true, as held by many physiologists, that the interchange of gases between the blood and tissues is not simply a matter of diffusion, but that the capillary walls have secretory power that causes them to play an active part in this process, we can see how in advanced cases of arterio-sclerosis, with many capillaries destroyed and others impaired, oxidation in the tissues may be imperfect so that lactic acid is not destroyed, but enters the blood and by reducing its alkalinity diminishes its capacity for transporting carbon dioxide which will accumulate in the tissues and cause a gradually increasing tissue asphyxiation. Or more probably the defective circulation consequent upon arterial changes impairs the nutrition of the glands and other tissues in which enzymes are formed, so that there is a scarcity of these necessary to complete the oxidation of lactic or any other acids which may be formed in the processes of constructive and destructive metabolism. This would probably explain the benefit that follows the prolonged use of alteratives such as the iodides of potash or mercury, which are known to have a very powerful influence on the glandular structures and nutritive processes of the body, although the manner in which they attain this end is still a matter of conjecture. The failure of the different alkalies when given by the mouth to influence the dyspnœa was a great disappointment because a diminished alkalinity has been shown to exist in this type of dyspnœa and there can be no doubt that the presence of acid in the blood, even in minute quantities, stimulates the respiratory center to greater activity. Bicarbonate of soda was given because of its great reputation as an atacid, especially in acidosis, potassium citrate because of its vaunted ability to render the urine and blood alkaline, as well as on account of the claim that in cases of lithæmia it promotes oxidation; citrate of soda for the reason that it is supposed to enter the blood as such where it is converted into carbon dioxide and sodium carbonate, thereby increasing the alkalinity of the blood and urine, and being eliminated in the urine as a carbonate. Sodium phosphate was the last selection made, and this was tried for the reason that physiologists have found that it re-acts towards carbon dioxide much in the same way as blood; so that formerly it was thought that phosphates in the blood plasma were an important factor in the evolution of the carbon dioxide. The chloride and lactate of calcium were also used, but failed to relieve the dyspnœa, in fact none of the preparations mentioned yielded the desired results. In stating that these different salts are supposed to have an alkalizing effect on the blood I am not giving my own opinion, but simply repeating the statement made by many writers; the data upon which their conclusions are based are not mentioned, but it would appear to be the opinion that because the alkalinity of the urine is increased, a similar change must take place in the blood. It is extremely doubtful whether alkalies or acids *taken into the stomach* ever materially affect the blood reaction. This opinion is based not only on such therapeutic

failures as those just mentioned, but also upon the general experience that such preparations as citric or lactic acid may be given in sufficient quantities to produce an extreme and even painful acidity of the urine without inducing any of the cardiac symptoms described by physiologists as accompanying the injection of even a minute quantity of acid in the blood. There is some provision of nature to maintain the blood reaction fairly constant during health, else there would be a continual variation with every meal according to the amounts of acids and alkalies ingested, and this protective influence cannot always be ascribed to a neutralizing combination with other food stuffs, as the result is the same when they are taken alone. It would look as though they entered into some combination in the lymph or tissues before entering the blood that rendered them ineffective, and that their elimination in the urine was accomplished by the decomposition of these compounds through the secretory activity of the kidney. The general acceptation appears to be that all alkalies are neutralized by the carbonic acid of the tissues and circulate in the blood as neutral bicarbonates without altering its reaction; and that while an excessive supply of alkali, by combining with the body acids, might be expected to render the tissues more alkaline, this is obviated by the rapidity with which the excretory glands remove the excess.

In this way there is more *available alkali* in the blood and tissues during alkali treatment, but it can be utilized in the neutralization of acids only when the carbon dioxide can be removed from the neutral salts. The clinician must therefore inquire from the bio-chemist and physiologist whether in some of our cases of arterio-sclerosis there may not be changes in the tissue cells that retard such chemical processes or make them impossible.

This question is permissible because observations both upon men and animals warrant the conclusion that the alkaline carbonates which are prescribed or taken in various waters for their antacid effect, when administered by the stomach do not exhibit the toxic effects of the sodium and potassium ions upon the cardiac muscle and other tissues because they are so rapidly excreted by the kidneys. Probably this is perfectly true in the healthy man, but is it equally so in disease? Vichy water abounds in alkaline bicarbonates and is used with impunity by the majority of people, yet there are others to whom the treatment at the spa is refused, and I am informed that this rule includes all cases of cardiac asthenia. Personally I have had a number of patients who, while using Vichy water on account of its excellent antacid effects, became depressed, languid, averse to muscular exertion, and dyspnœic. Improvement followed rapidly upon discontinuance of the water, and upon its resumption the symptoms soon returned. I have seen symptoms of cardiac dyspnœa with reduced blood pressure occur in patients who were taking from twenty to thirty grains of potassium iodide daily, while a change to one of the preparations of calcium iodide or hydriodic acid obviated any unpleasant symptoms. This is the exception rather than the rule in patients suffering from arterio-sclerosis and there do not appear to be any special physical signs in the heart or blood vessels that aid in identification of the susceptible group. Hence the question has to be asked whether the interchanges that normally take place between chemical substances within the body are not facilitated by some functional activity of the tissue cells, and whether apparent discrepancies between the conclusions arrived at from experiment or observations of healthy subjects and the results obtained by the clinician at the bedside are not due to changes in the tissue cells. In regard to treatment this is of importance because an affirmative answer would imply that the practitioner must not content himself with any halfway measures, as for example an attempt to neutralize excessive acidity, but must also endeavor to restore the function to the diseased tissue cell or in some way compensate for it. We have an example of the latter in the treatment of myxœdema by the administration of thryroid gland preparations.

Outside the body oxidation is accelerated by the presence of an alkali, and consequently, alkalies have been administered in the hope of producing a more perfect metabolism; but, possibly on account of the rapidity with which the excess of alkali is eliminated or transformed into the neutral bicarbonate, the laboratory experiment does not find its homologue within the human body, at least the reported observations upon this subject are very contradictory. We expect that the true treatment of such cases must await the telling of the secrets of metabolism and organo-therapy, for the human body is neither a retort nor a test tube, but a whole chemical factory. At present I am treating some cases with thyroid extract, as that gland is reputed to play an important role in metabolism by promoting the oxidation of proteids, but the results are too imperfect for publication at the present time.

There is another feature regarding the dyspnœa of arterio-sclerotic patients that is worthy of consideration. Nervous irritability is one of the characteristic symptoms of arterio-sclerosis. These sufferers undergo a change of disposition, little things annoy them, and the consciousness of the fact annoys them still more, their emotions are more easily influenced than formerly, and either joy, sorrow or mental effort may induce an attack of dyspnœa. It seems to me that in many instances the respiratory center participates in this cerebral hyperaesthesia, so that normal quantities of carbon dioxide or any of the acid products of metabolism may more readily induce increased respiratory movements, in other words, that in some cases the cardiac dyspnœa may be due more to hyperaesthesia of the respiratory center than to diminished alkalinity of the blood. Recognition of this condition explains the dyspnœa that awakens the patient from his sleep, because it is generally admitted that this hyperaesthesia is the result of impaired cerebral nutrition; and during sleep the fall of cerebral blood pressure and the shrinking of

the brain substance in consequence of vascular relaxation throughout the body generally still further impairs the nutrition and increases the irritability of the respiratory center so that it responds to a stimulus from blood in which, as metabolism is diminished at this time, there is no reason to expect excess either of carbon dioxide or any other form of acidity. Sometimes the condition is relieved by a combination of digitalis or strophanthus or caffeine with one of the nitrites, as it would seem that a combination of a vaso-dilator and cardiac stimulant is more effective in maintaining an adequate circulation through the sclerosed vessel, thereby removing the irritability of the nerve centers arising from impaired nutrition, but in a very large number of cases the patient's distress is not relieved until small doses of morphine, one thirty-second or one twenty-fourth of a grain, every eight hours, are added to the mixture.

The most complete discussion of this subject with which I am familiar can be found in a composite article by Lewis, Ryffel, Wolf, Cotton and Barcroft, entitled "Cardiac and Renal Dyspnœa," that was published in *Heart,* Vol. 5, No. 1, 1913. This investigation consisted in a most elaborate examination, at the bedside and in the laboratory, of fifteen patients; and although ten of that number had pleural effusion, ascites or general dropsy, still, in the judgment of these gentlemen, such mechanical obstructions to respiration were not sufficient to explain the degree of dyspnœa. This is perfectly possible but, as it is a question of individual judgment, those readers who have not seen the patients cannot help wishing that such respiratory embarrassments had not existed. It is interesting to note that abnormal fullness of the veins is mentioned as one of the clinical features in thirteen out of the fifteen cases, no reference being made to the condition of the veins in the other two patients; and in only two of the whole series of fifteen cases was cyanosis well-marked, in the others it was very slight or entirely absent. In only one case did the haemoglobin fall below 75 per cent., and in the others it ranged from 80 to 102 per cent. In all of the patients there was some degree of acidosis or diminished alkalinity of the blood. They express the opinion "that as no evidence of the formation of an abnormal amount of any organic acid is to be obtained from examination of the urine, so this acidosis appears to be another instance of the same phenomena of the retention of acids in excess of bases which is shown during residence at high altitudes." The thought, therefore, was perfectly natural that if these forms of cardiac dyspnœa resemble that of high altitudes some benefit might be expected from a line of treatment calculated to produce changes in the blood similar to those that occur during the process of adaptation. This consists in an increase of red corpuscles to seven or eight millions per c. m. m., at first probably by concentration of the blood, but ultimately from increased activity of the blood-forming organs. During the establishment of adaptation the pulse rate is accelerated so that in the rarefied atmosphere adequate oxygenation will take place by blood abnormally rich in haemoglobin circulating more rapidly. Most of my cases have shown haemoglobin to be present only in the proportion of from 65 to 85 per cent., and under the administration of arsenic and iron there was some improvement, but not enough to awake enthusiasm. A review of the tabulated statement of the gentlemen already mentioned is no more encouraging, as they report cases of orthopnœa in which there were five and even six million red corpuscles to the c. m. m., with from 95 to 102 per cent. haemoglobin.

The failure of alkaline treatment and inhalations of oxygen to relieve the dyspnœa, together with the co-existence of full veins, abundance of red corpuscles with high color index, and the absence of cyanosis still farther impresses one with the ideas already mentioned that in arterio-sclerosis there is some flaw which prevents the tissues from utilizing the oxygen supplied to them, and suggests diminished function owing to degenerative changes in the vascular endothelium as a possible cause. For many years evidence has been accumulating that these cells have distinct chemical functions, and the recent work of Hooper and Whipple, who have shown by experiments upon animals whose livers had been excluded from playing any part in the process, that vascular endothelium in common with the mesothelium of the pleura and peritoneum have the power of transforming haemoglobin into bilirubin by splitting off the iron from the former demonstrates conclusively that these cells can no longer be regarded only as having a mechanical function, such as affording protection to different surfaces or forming a system of tubes for conveying the blood throughout the body, but that they are active participators in the chemical processes of the body. Possibly the foregoing remarks may cause some amusement to the physiologist or bio-chemist, because the problems are so self-evident to him, but on the other hand, the clinician not infrequently smiles at the dogmatism of laboratories and admires the unblushing assurance with which at some subsequent period, a stoutly defended theory is discarded as absolutely untenable.

Nevertheless, on this occasion I am willing to be the scapegoat if the sacrifice makes plain the necessity for a closer association between the different departments of medicine, and emphasizes to our undergraduates that anatomy, physiology and chemistry are not studies preliminary to medicine which may be neglected so soon as a diploma is obtained, but that they, too, are progressive subjects, that the anatomist, physiologist and chemist have still much to learn, and that as the clinician is the man who must utilize and turn to practical account the discoveries of these other gentlemen he can only hope to do so if he keeps himself informed regarding the work that they are doing.

The scope of medicine is too great for a man to do this by his own effort; it is only possible for him to attain this end when he is so situated as to confer at will with the individual observers or to hear from them at intervals a summary of the progress made in their departments.

SYPHILITIC ARTHRITIS.*

By LEONARD W. ELY, M. D., San Francisco.

Morbid processes in joints are comparatively simple in their essentials. While there have been extremely involved and complicated classifications made at various times, of bone and joint inflammation, yet the fundamentals of these classifications are comparatively simple. In order that you may know how to apply what I am saying to the ordinary types of disease, I would call your attention to the fact that the type of inflammation which we are going to speak about mostly tonight has an analogue, known by various authors as atrophic arthritis, as infectious arthritis, as proliferative arthritis, and as rheumatoid arthritis.

If the marrow and the synovial membrane be exposed to an irritation, they respond in a certain way. If, for instance, we have a tubercle in the marrow of the end of the bone, spreading and involving more or less of the marrow, we will get an atrophy of the bone. If the disease breaks through and involves the joint, we will have a proliferation of the synovial membrane. Instead of forming a comparatively flat, smooth membrane, it will be thrown into folds, and what is known as villous arthritis or synovitis will result. If the gonococcus be the invading organism, practically the same thing takes place. Typhoid and pneumococcus joints have the same pathological fundamentals, and syphilis precisely the same. Thus, a syphilitic inflammation in the end of the bone causes a rarefying osteitis, an atrophy of the bone trabecula, and a villous condition of the synovial membrane. As a result of this, the synovial membrane will encroach upon the cartilage from its side, displace the cartilage, tend to spread out at its circumference, and also over its surface, while at the same time the disease in the marrow not only will cause a rarefying of the bone trabeculae but will also shut off the nutrition of the cartilage and perforate it from its under surface.

Cartilage, when it is diseased, is no longer immune to morbid processes, but is perforated by these syphilitic granulations in the marrow. There is nothing radically different in any of these diseases, in the reaction of the bone, of the bone marrow, of the synovial membrane. They all cause practically the same changes. Now, pathological identity means identity of symptomatology and physical signs. Practically, the symptomatology of all these diseases is the same. In a general way, each has its peculiar points, but there is nothing absolutely characteristic about the course of any one of them, nor is there anything characteristic about the symptomatology or of the physical signs. Hence, it follows that tuberculous arthritis cannot be differentiated from any other member of the group by the clinical symptoms.

I was taught in my younger days that syphilitic arthritis was so rare in children that it could practically be disregarded as far as clinical work was concerned. On the contrary, the more you look for it, the oftener you find it. If a joint be inflamed for any reason, the first, the peculiar characteristic of the inflammation, as with the inflammation of any other organ of the body, is a disturbance in function. If an inflammation exists in the kidney, the kidney function suffers. If an inflammation be present in the heart or in the lungs, the same thing happens. If an inflammation, no matter what its cause, be in the joint, motion is usually the first thing to suffer.

Tuberculosis starts in the epiphysis, not so often as syphilis, but very often; and either can start in the synovial membrane and later involve the marrow. Syphilis usually involves the shaft and rarely the ends of the bone. If the shaft and the end of the bone are both involved, the diagnosis of syphilis is probable. There is no known way of diagnosing syphilitic arthritis except, I believe, by the therapeutic test. If we know that the patient be syphilitic, we have a strong indication, but a tuberculous joint may exist in the syphilitic individual, as the syphilitic joint may exist in a tuberculous individual.

Given a case of a supposed syphilitic arthritis, the joint will usually be swollen. The swelling may be due to the thickening of the synovial membrane, to fluid in the joint, or to both. Muscular atrophy and muscular spasm may be present or absent. One joint may be involved or several joints. Various writers describe peculiar forms of the disease and speak about the synovitis of hereditary syphilis. Sometimes the disease does exist as a comparatively painless synovitis, but the marrow may be involved as well.

There are four frequent forms that the disease may present: One, synovitis; another, what is known as true arthritis—inflammation of the synovial membrane and of the bone end itself; another, the peculiar multiarticular form of arthritis which simulates that caused by the diplostreptococcus of Rosenow. Then there is a late form of the disease known as the Charcot joint.

Pain may be prominent, or it may be absent. The peculiar painless joint is suggestive of syphilitic synovitis. Tuberculosis usually has a good deal more pain than syphilis, but not always so. If only the synovial membrane be involved, tuberculosis may also be accompanied by very little pain. As a rule, the disease is slower and less destructive than is tuberculosis. If it has existed for a long time, if it has perhaps disappeared for a while and then returned, and if it is not painful, the indications are that it is not tuberculosis but syphilis. The diagnosis comes down as a rule to one of five or six morbid processes; the first, tuberculosis; the second, syphilis; the third, gonorrhea; the fourth, typhoid arthritis, which is extremely rare; the fifth, diplostreptococcus of Rosenow; and the sixth, trauma. In the X-ray of all these diseases, we may find practically nothing but a thickened synovial membrane; but as a rule, we will find an erosion of the cartilage, and a rarefaction of the bone. In lues, the process is apt to be on the joint side of the epiphyseal side; in tuberculosis, on the shaft side. Tuberculosis usually affects the synovial membrane and the marrow. Gonorrhea usually affects the periosteum and the synovial membrane, rarely the marrow.

* Read before the Alameda County Medical Association, January 18, 1916.

Lues, as a rule, affects the marrow and the periosteum, not so often the synovial membrane. The Wassermann test helps, but it is not conclusive.

The weak point in all our work on syphilitic bone and joint disease is that we are compelled practically to accept the diagnosis of lues when the disease disappears on the administration of antisyphilitic drugs. It is no proof. We know it, yet it is all the evidence we have at hand.

The good old rule never to operate upon one of these tuberculous joints until it has been proved nonsyphilitic by administration of antisyphilitic remedies is a very safe one. No one seems to be immune from this mistake. I stand here emphasizing this to-night, and I have no doubt that within two or three months I shall make the same mistake.

The only way to solve this problem is not by looking at one joint, but by looking at the patient as a whole—going over him from head to foot, thinking what the disease may be and making our diagnosis by exclusion.

The treatment of syphilitic arthritis is simple. If the Wassermann test is positive, use salvarsan followed by mercury and the iodids. If the Wassermann test is negative, and you are not very sure of your diagnosis and wish to be on the safe side, use mercury and the iodids, mercury especially. The iodids do not seem to have very much effect on these syphilitic joints. If the Wassermann test is negative and we are very suspicious of syphilis, then administer salvarsan followed by mercury. It is rather remarkable that very often these joints will improve on the administration of mercury for a while, and then will come to a standstill. A dose of salvarsan seems to hasten things. Then it is well to go back to mercury. Strange to say, immobilization has no effect upon them whatever.

It is almost diagnostic of tuberculosis that when the joint is put at rest the improvement starts in. Syphilis does not respond to immobilization, but on the contrary, often grows worse. Plaster of paris and braces do no good, not even as adjutants. It is interesting to see in some instances how an apparently hopelessly distorted joint with its architecture destroyed, will re-form under appropriate treatment.

Syphilis can affect fibrous tissue, red marrow and yellow marrow, or synovial membrane, whereas tuberculosis affects nothing but the lymphoid marrow and the synovial membrane. Syphilis does not have any distinct boundaries. You cannot say when a child, three or four years old, is brought to you for treatment, "This cannot be a syphilitic synovitis, because congenital syphilis in the child appears in such and such a week, and is steadily progressive or tends to recover, or what not." You cannot bound the disease or its appearance in the joint by artificial rules. There is no known way that you can tell syphilitic joint by appearances. Do not think that if an inflammation comes at a certain time, syphilis is barred out. It is never barred out. Syphilis is never out of the question. Do not always depend on your periosteal thickening. Look for it. If you get it, you are pretty sure; but if you do not get it, do not rule out syphilis.

As I said before, this all must be taken with a grain of salt. We cannot take a joint into a laboratory, and say it is or is not syphilitic. In tuberculosis, we can remove fluid from a joint and inject it into a guinea pig. If the guinea pig has tuberculosis, then the joint is proven to be tuberculous. If the guinea pig is healthy, the joint was not tuberculous. If we remove a piece of tissue from a tuberculous joint, and find characteristic tubercles in it, we say that it is a tuberculous joint. We cannot do that with a syphilitic joint. The best we can do is to say, "This may be syphilis." We have no infallible guide. As a rule, the spirochaete are not to be demonstrated. The only rules that we can say we possess are always to be on the lookout for syphilis when we find a slow inflammation in a joint; always to be on the lookout for it when we find a rapid inflammation in the joint; never to lose it from our minds, to remember that in its florid stage, the joint may be swollen and the bone rarefied, that cartilage may be eroded at its margin, perhaps eaten out in its center. It is all more or less vague. The vagueness disappears when the arthritis clears up under syphilitic treatment. The vagueness comes in its confusion with the other diseases, and the vagueness disappears if the disease clears up under antisyphilitic treatment.

Discussion.

Dr. Milton: Dr. Ely has left little else to be said on the subject. You will have to remember in treating joints that joints are subject to syphilis, and in large clinics where this subject is gone into pretty thoroughly, it is found that a large percentage of the joints are syphilitic, as high as eight per cent. in some clinics. So we have to remember that the thing is about us, it is with us.

Years ago when I went to college, they used to tell us that syphilis and tuberculosis were incompatible; if you got one, you could not get another, and vice versa. We know now that it is not so. Now, we have to remember that joints can be syphilitic, and we have to be on the lookout for it.

Dr. Buteau: This is too good a paper to let go undiscussed. If it is not more discussed, it is simply because Dr. Ely has thoroughly covered the subject, and because we recognize his experience and feel a little timid in bringing forth any arguments that might possibly suggest themselves to us.

I remember many times when I have had opportunity of visiting some of the larger clinics in the East that I would come back again and again, and say, "Well, they do not have the kind of cases back there that we have here—at least, the kind of cases that I have. My bad cases seem to be worse than any of the cases I see in some of the bigger clinics that are handled by masters in our profession." After a time, I began to realize that it was the way they handled their cases. They did not seem so serious because of the intelligent way in which they were approached. The operations that were awful to me were simplified often and made easy by these master men because they arranged their field so neatly. The rest of the work was readily done. And I was impressed here to-night by the way the Doctor approached his subject and the concise manner in which he presented and simplified it, and to my mind made it appear that these joint troubles were not such complicated cases to master after all.

There is only one question I would like to ask: Dr. Ely has mentioned six different kinds of troubles or affections with which syphilis of the joints might be complicated or confused in making a diagnosis. I wonder if we might not add malignancy. I know that is more rapid in its development of symptoms and yet in my experience again and again have I called it into question in making the diagnosis.

Also another question I would like to ask the Doctor: "Would it not be well at times to assume the diagnosis to be tuberculosis instead of syphilis and possibly to put the joint at rest and note the results of that treatment, as well as to assume that it was syphilis and put the patients under the treatment of antisyphilitic remedies?"

Dr. Ely: Dr. Buteau's remarks are well taken. Malignancy is very frequent at the end of the bone and is difficult to distinguish from syphilis. As a rule, a malignant disease does not involve the joint. There are joints that are so typically tuberculosis that you will immobilize them and will not give antisyphilitic treatment. That is correct, and yet, if you do that, always be on the lookout to prove that you were wrong. Always look for bony thickening, for an enlarged spleen, for something that may suggest syphilis. All these diseases are liable to secondary infection. Tuberculosis, especially, runs its regular course. If proper treatment is not administered or in spite of proper treatment, the diseased area may break down and a secondary infection may be added to it. If a secondary infection be added, the diagnosis is easy. As a rule, the sinus in typical, but do not think that you can make an invariable diagnosis from the appearance of the sinus. This is another error that we have just struggled out of after many years. Very often there is the typical pale, flabby look of a tuberculosis sinus. A gonorrheal joint, as you know, may at any time become secondarily infected; so may a typhoid joint, a pneumococcus joint. Some are more liable to secondary infection than others.

What I have given you is not only the result of laboratory work, but it is a struggling out of a confusion, a graduation from an idea that especial acumen could be cultivated to diagnose these joints. We used to be taught that they were perfectly simple. If a patient was brought in with a chronic inflammation of a joint, it was assumed to be tuberculous. There was practically no discussion. It was put in plaster of paris, and it went on in its treatment for years. I would make a diagnosis of tuberculosis or gonorrhea or syphilis. Sometimes I would strike it right and sometimes I would strike it wrong, and then I was crestfallen. I thought it was my own stupidity. At Roosevelt Hospital, where we had quite an active clinical service and quite a friendly rivalry, when anything interesting like that came along, each man would make his diagnosis and would bet on it, and the youngest man in the clinic would be just as liable to upset the oldest as the oldest to upset the youngest. It seemed to be a mere matter of chance. Then I set to work with these joints that had been removed and I put them under the microscope. I collected their histories, found out the diagnoses that had been made upon them by various men, the operations that had been done by various men, and I found that about one-third of them had been erroneously diagnosed. In the vast majority of instances, perhaps one man showed a little better average than the others. Those joints included the work of some of the biggest men at that time in New York, some of them were my own joints. From that it soon became evident to me that the diagnosis could not be made on the usual lines. Then from that and a study of the pathology of joint tuberculosis, it dawned on me how the diagnosis should be made. It was not to be made by any general appearance of the bone, not by any particular, you might say general, appearance of the synovial membrane, not by any particular appearance of the marrow in gross. Submit a piece of a joint to a pathologist. He will look at these various tissues, and if he find a tubercle in the synovial membrane or in the marrow, he will say that it is tuberculosis. If he do not find a tubercle, he will call it chronic inflammation—non-tuberculous. The features of both are the same, and the only point that microscopy did for the diagnosis was to say "chronic inflammation," tuberculous, or non-tuberculous. If a man cannot make it in the clinic from an inspection of the joint, and the pathologist cannot make it except from the presence or absence of the tubercle, how can we make it? From the X-ray? Well, take your X-rays. Take the best men in X-ray diagnosis. Have them put their diagnoses down, have them follow their cases along. You will find the X-ray man cannot do it. The X-ray features are all the same. History? The man who will go deepest in the history will guess right more often. If he pulls up the patient's trousers, finds the knee swollen, and says chronic rheumatism from the appearance of the joint, he will miss it most often. Let him sit down and enquire into the patient's health—how many times he has had a Neisser infection; if he ever had syphilis; if he ever had pneumonia, typhoid fever; how many joints were involved and in what order; did the disease come on suddenly or slowly? Let him ask, "Are your parents alive?" "What is your occupation?" The deeper one goes into the history, the more often one will guess right. A thorough physical examination must be added, and the usual laboratory tests. Then one is in a position to guess right in the majority of instances. If one will then on top of hard thorough work and careful examination of his patient, recognize the fact that one cannot make a positive diagnosis, and if one will keep one's eye open to the possibilities of error, one will do as well as is possible.

THE TONSILS AS A FOCUS ON INFECTION.*

By JOHN MACKENZIE BROWN, M. D., Los Angeles, Cal.

It has long been recognized that the tonsil plays an important role along with many other structures of the body, as a possible site of focal infection. But it has been only during the past decade that sufficient emphasis has been laid on the relative importance of the tonsil in comparison with other focal lesions in the production of morbid processes in other locations, and general systemic diseases. The work of such men as P. K. Brown, Billings, Shaumbaugh, Rosenow and others, has demonstrated conclusively that many pathological conditions of obscure origin are due either directly, or indirectly, to bacterial or toxic absorption from pre-existing or active processes in the tonsil.

The question naturally arises, why are the tonsils so closely related to systemic infection? It is because they are probably the most often infected of any of the possible sites where pathogenic organisms may be harbored. And this susceptibility to infection appears to be mainly due to their anatomical structure and position.

The tonsil, situated as it is near the commencement of the alimentary tract, is surrounded at all times by a flora of both pathogenic and non-pathogenic germs of the mouth. The crypts pass deeply

* Read before the annual meeting of the California State Medical Society, Fresno, Cal., April 20th, 1916.

into the lymphatic tissue proper, in various directions, forming pockets or recesses in which microorganisms find warmth and moisture which plus lowered tissue resistance means multiplication and invasion. Again, many of these tonsillar culture tubes are long, narrow and branched, these facts together with the inclination of the crypts, favor stasis. Strictures become comparatively easy following even slight inflammatory conditions, thus producing pockets or foci where incubation may continue. In other words, the tonsil after a thorough infection becomes a veritable culture tube for the bacteria that inhabit it.

A word as to the bacteriology of the tonsil with respect to focal infection seems necessary to show, if possible, the etiological relationship between disease existing here and other acute and chronic manifestations. Recently Rosenow has published an extensive article on the elective localization of the streptococci, showing how focal lesions both acute and chronic, are responsible for many and varied systemic conditions. He states that foci of infection afford opportunity for bacteria to grow under varying degrees of oxygen pressure, and in mixed culture, both of which have been shown to cause changes in virulence and other properties of bacteria. He cites such systemic infections as rheumatic fever, appendicitis, and ulcer of the stomach, following the acute symptoms in follicular tonsillitis being caused by the streptococcus group of organisms. Rosenow's work was based upon animal experimentation, and he has apparently proved his results conclusively, by the finding in the focus, streptococci having elective affinities for the same structures in animals.

Davis of Chicago, by careful laboratory investigations of tonsils removed for various systemic disturbances, found hemolytic streptococci in twenty-eight cases of arthritis. In one case in association with pneumococci, in another with the streptococcus mucous. In ten cases of nephritis, nine showed hemolytic streptococci, pure or predominant in the crypts, so that these serve as examples of the predominance of the streptococcus group. Among other infecting organisms may be mentioned staphylococci, b. influenza, b. diphtheria, b. coli cummunis, and b. tuberculosis, the latter, it has been demonstrated by Wood, and others, can be made to invade and pass through the tonsil without producing tuberculosis of the tonsil itself.

Again Smith and Barrett have shown that the endamoeba buccalis has been found in the crypts and tonsillar tissue of extirpated tonsils, and they state that this organism is not only responsible for Riggs disease (pyorrhea alveolaris), but has many general manifestations as well.

The tonsil, I have emphasized, is one of the commonest containers of focal infection, and it necessarily follows that many acute and also chronic disturbances attributed to focal infections in general, applies to the infection of this organ, except with greater frequency. Inflammation of the tonsils has long been found to be associated with acute rheumatic fever, but not until the researches of Poynton and Paine was this relationship definitely understood as that of cause and effect, and now as Halsted of Syracuse states, clinicians and bacteriologists alike are agreed that the most common habitat of the micrococcus rheumaticus of Poynton and Paine is the tonsil. It may lie quiescent for months or years until the lowered vitality of the individual invites invasion, and then migration to a seat of election may occur in the joints, tendon sheaths, valvular endocardium, pericardium, or the central or peripheral nervous system.

Rosenow has produced appendicitis experimentally in animals by the injection of tonsillar extracts. He has produced in the same manner other lesions from various strains of streptococci.

In connection with chronic disease, the role played by focal infection from the tonsils seems quite as important, but research along this line has been still more recent. A few lesions, however, may be mentioned as at least influenced, if not caused, by intoxication or bacterial invasion from the tonsil. Probably heading this list would come the chronic arthritides upon which so much work has been done of late. I will admit mostly, in an empirical way, but in a review of the literature on this point, I find reported many cases of chronic deforming arthritis that have been greatly improved by enucleation of the tonsil, which in most cases showed the presence of the streptococcus hemolyticus.

Not only does the focal lesion in the tonsil seem to be responsible for the many chronic conditions, but writers have laid stress on the slow chronic intoxications or which really in many cases would come under Adami's group of sub-infections, which are undoubtedly responsible for many of the premature degenerative changes seen in many individuals and which produce effects varying from chronic indisposition to early degenerative changes in the cardiovascular and nervous systems and the chronic nephroses. Kyle, of Philadelphia, emphasizes asthma as a disease caused by toxic absorption from the tonsil, and Tschiassny reports a case of recurrent bronchial asthma in which removal of the tonsils resulted in a cessation of the attacks. Beck, of Chicago, also states that exophthalmic goitre undoubtedly is caused directly, or through alterations of the internal secretions by toxicity due to focal lesions of the tonsil. Again, to quote Billings, who says: "There is no doubt that the insidious, slow, degenerative processes which occur in many patients who arrive at the meridian of life are due to slow intoxication from chronic focal infections variously located, one of the most frequent of such focal infections being the tonsil."

It is not my intention in a paper of this length, to show how many different and varied conditions might hypothetically be caused by diseased tonsils, but how intimately some of the more important conditions are related in this way.

In any given case of acute or chronic disease thought to be due to tonsillar infection, after due consideration of other possible foci, how shall we arrive at a conclusion as to whether or not the tonsil should be removed?

In a case of acute tonsillitis complicated by an

intercurrent and seemingly directly associated condition, such for example as acute cardiac or renal disease, acute rheumatic fever, Sydenham's chorea, etc., the rational procedure seems a tonsillectomy.

In the case of chronic disorders the appearance of the tonsil often lends valuable information, the septic tonsil is frequently dark red in color, the crypts may be sealed over and superficial abscesses may be seen on its surface, the anterior and posterior pillars may be unduly adherent. Pus with cellular detritus, the result of previous tonsillar inflammation, may be squeezed from the crypts. The condition of the neighboring structures often gives additional evidence, the pillars may be of a deep red color, edematous, and lymphoid patches may be found on the posterior wall of the pharynx just back of the tonsil. The size of the chronic tonsil has nothing whatever to do with its relation to septic foci, in fact, the hypertrophic tonsil seems to be less a factor in the production of these various manifestations than the smaller imbedded tonsil. Again, in many cases what appears to be normal-looking tonsils when seen in situ, after enucleation I have found to contain pockets of pus hidden from the exterior.

In some cases I believe there is no way of telling whether or not the tonsil is infected so that if the external evidence does not seem sufficient to warrant removal for definite and recognizable lesions present, I strongly suggest the consideration of their removal if chronic infection or intoxication, seemingly points to this situation as the cause of trouble, after the careful exclusion of other possible foci. At the present state of our knowledge, regarding the connection between focal lesions, and metastatic infection and intoxication, I am inclined to take the empirialistic view, and look with suspicion on any tonsil found in an adult having unexplainable chronic symptoms. Here, I cannot emphasize too strongly, the necessity for thorough enucleation which is the teaching set forth by Billings. To leave the smallest portion of tonsillar tissue, means possibly a focus tightly sealed, when scar formation is complete, thus defeating our primary object. This fact explains the disappointing results seen in some cases, where a supposedly complete tonsillectomy did not give the promised relief. So the thought I want to leave with you, in this connection, is: Be just in judging; what by some is termed indiscriminate tonsillectomes, and study your series of cases which you count as failures, with this point in view. If this is done I think you will agree with me when I state that in conditions where you lay the fault to the tonsil, a thorough and careful enucleation is the only safe procedure to follow.

I wish to review briefly a few cases, although our highest hopes in every particular were not realized, however, in the main our efforts have been encouraging, and our course of action seems justifiable.

J. P., boy, age 7. Has had a superficial keratitis of the right eye for four years, during which time there were recurring attacks of acute tonsillitis, and associated with these attacks the eye condition became worse. The case was under the treatment of oculists during this time, but however the condition remained unchanged. The tonsils on examination were found diseased and their removal advised. One week after the tonsils were enucleated, the eye began to clear up, and was apparently normal in three weeks, and has remained so for the past four months.

A. P., age 22. Patient entered hospital complaining of headaches and weakness. Blood pressure 200 m.m. Urine showed considerable quantity of albumin with hyaline, and granular casts. Later he developed uraemic symptoms and at times became delirious. There was no history of acute nephritis or acute infectious disease, but a history of recurrent attacks of acute tonsillitis; on examination the tonsils were enlarged and clearly septic. Enucleation was advised, and done. The condition at this time was almost hopeless, as the kidney function was greatly impaired, and the patient's general condition was becoming rapidly and progressively worse. The results were quite unsatisfactory. The general condition did not improve, and the patient died three months later.

H. M., age 25. History of recurrent attacks of acute tonsillitis, the tonsils were supposedly removed three years previous. Acute nephritis a little over two years later, following acute tonsillitis. Albumin and casts have been constantly found in the urine since. Examination showed a small stub of one tonsil which was clearly septic, this was removed. His general condition commenced to improve immediately, also there was very much less albumin and fewer casts in the urine. At present time, a year and a half after the operation, he still has a trace of albumin and an occasional cast, but his general condition is excellent.

H. S., age 54. History of repeated attacks of tonsillitis all her life. At the age of 40 arthritic symptoms developed commencing in the meta carpal and phalangeal joints, the condition advanced rapidly with deformity and severe pain until a few years later she was a chronic invalid from arthritis deformans. The arthritic condition was always more painful during an attack of tonsillitis, which fact, along with local evidences in the tonsils seemed sufficient grounds for their removal; after enucleation the joint condition became stationary and the pain began to subside, she was able to do her own house work in six months later. At present, four years after operation, she is in good health, and comparatively free from joint disturbance.

M. S., age 22. During an attack of acute tonsillitis she developed an acute nephritis which gradually improved, but the urine continued to show the presence of red blood cells, granular casts, and a trace of albumin, and during the next six months there were several acute attacks with increase of all the kidney symptoms. The tonsils in the patient were chronically inflamed, and after the careful exclusion of all other possible foci, their removal was advised. Four years have elapsed since enucleation; the urine still shows a few red blood cells and granular casts, but the acute exacerbations have entirely ceased. The general condition is excellent.

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Discussion.

Sanford Blum, M. D.: The role played by infective foci in the etiology of endocarditis seems to be regarded as a new discovery. It is not very new. It has merely received wider recognition since it has been presented by men in more prominent positions; but the association has long been known and recognized. In 1902 I presented for a Master's degree, at the University of California, a thesis entitled "The Etiology of Endocarditis with Especial Reference to Bacterial Agencies." This paper was published in American Medicine, January 17, 1903, Vol. 5, No. 3, page 94 seq. In it I stated that "all bacteria pathogenic to the individual may cause endocarditis under the proper conditions. There must be a locus minoris resistential and the bacteria must be in the blood. They may enter the circulation from various sources—from an abscess of the foot (Winge's case), from the septic womb, from the intestines." But no single case of endocarditis has, so far as I am aware, been positively proved to have emanated from infected tonsils. Many cases of endocarditis have been discovered after tonsillitis, but post hoc does not prove propter hoc in these cases.

It has been stated to-day that when endocarditis exists, removal of infected tonsils (assumed to be the source of infection), should halt the process and prevent further destruction from the endocardium. Even if it should be assumed that the tonsils were the original focus from which the endocardium became infected—it seems illogical to conclude that, after the endocardium has become infected and a metastasis established, removal of the distant focus would check the process. Nor has tonsillectomy had this beneficent result in endocarditis cases which I have seen.

In a paper, "The Proper Position of Tonsillectomy in Pediatrics," read before the California Pediatric Society April 22, 1915, I cited cases of endocarditis beginning subsequent to tonsillectomy. Koplik (American Journal of Medical Sciences, July, 1912), reports similar observations. J. Herbert Young (Boston Medical and Surgical Journal, September, 1915) has published reports of 21 cases of tonsillectomy which he had under observation for two years following operation. Twelve of these cases had endocarditis before tonsillectomy, while 17 had endocarditis after tonsillectomy. In one case acute endocarditis was present, five days after operation in a child, whose heart had appeared normal before operation. In a second case endocarditis developed two weeks after tonsillectomy. The above quoted observations appear to imply that tonsillectomy not only does not prevent or check endocarditis, but may even be a factor in its causation.

Young's observations throw light on the relation of tonsillectomy to chorea. Of his 21 cases, 12 had chorea prior to tonsillectomy; 17 had chorea subsequent to tonsillectomy. Every case in which chorea was present before tonsillectomy, had from one to four attacks in the two-year observation period, subsequent to tonsillectomy, and five additional cases occurred after the operation. In the paper referred to above, "Proper Position of Tonsillectomy," I cited similar observations. These records indicate that the theory that tonsillectomy is a cure or preventive of chorea is false.

It is my privilege to acquaint Dr. Gundrum with an authentic case of endocarditis emanating from intestinal infection. In 1898, while serving in Escherisch's clinic in Graz, I saw an infant, which had pyocyaneus enteritis. There developed pyocyaneus septicemia and pyocyaneus endocarditis. Cultures of pyocyaneus bacilli were obtained from the feces and blood of the infant, and after its death, the same bacilli were identified in the verrucosities on the endocardium. Cultures obtained from the blood, were injected into the ear vein of a rabbit, of which I had lacerated the endocardium with a probe, introduced through the carotid, and pyocyaneus endocarditis developed in this rabbit. This case was published in the Centralblatt für Bakteriologie, Parasitenkunde, und Infektionskrankheiten, 1898, No. 25.

The tendency to condemn the tonsils, generally, as foci of infection should be criticised—as the theoretical benefits credited to tonsillectomy are not borne out by the facts. Simpson (Jour. A. M. A., April, 1915) observed, in Girard College, that scarlet fever and diphtheria occurred in the same proportions in tonsillectomized and untonsillectomized children. He reports also that a boy whose tonsils had been enucleated became a diphtheria carrier. There is at present (as I was to-day informed by Dr. O'Neill) in the San Francisco Isolation Hospital, a diphtheria carrier, who became such a year after tonsillectomy. It may be asserted that failure of tonsillectomy to fulfill its promises is due to faulty operation, but it is with tonsillectomy as it is performed that we have to deal.

FOCAL INFECTION INTESTINAL INVOLVEMENT.*

By F. F. GUNDRUM, M. D., Sacramento.

The development of a general invasion such as septicæmia or pyæmia from an originally localized bacterial infection has been a matter of medical observation and knowledge for many, many years. It has been but recently, however, that the relation of a once acute and now quiescent local infection to disease conditions in widely separated portions of the body has been recognized. Lubarsch has shown that a focal infection may remain dormant for a long time until some often extraneous event may bring about a new activity. The pioneer work in this field has been done for the most part by Billings, Rosenow, Davis, Jackson and others in this country. Their researches have shown that a local infection may become sufficiently chronic that it causes but comparatively slight disturbance in its own neighborhood, but may still give off active bacteria or toxins to the blood stream and produce lesions in remote structures. Further that organisms of the Strep-pneumo group may under some circumstances of oxygen supply undergo transmutation in type and pathogenicity.[1] Many different organisms have been isolated from lymph glands which lie upon the lymph channels

* Read at the Forty-fifth Annual Meeting of the Medical Society of the State of California, Fresno, April, 1916.

draining chronically inflamed joints, Streptococcus, B. mucosus, B. Welchii, Diphtheroids, etc.[2] The greatest majority of these sites of focal infection whose pernicious activity has been proven lie in and about the head, i. e. teeth, tonsils, accessory sinuses, etc. A less number have been found in the respiratory and genito-urinary systems.

There has been up to this time relatively very little written concerning the role of the gastro-intestinal tract as a location for these indolent cryptic infections. It is most likely that a healthy gastro-intestinal mucosa offers very little opportunity for the development of this sort of difficulty. Under some conditions, however, foci may be established which could result in absorption. Billings,[3] in enumerating various focal infections likely to prove dangerous to joints, heart, kidneys, etc., mentions chronic ulcers of the stomach or bowels; chronic appendicitis ("damage chiefly to cardio-vascular apparatus"), and cholecystitis and cholangitis ("damage to cardio-vascular apparatus and kidneys"). A chronic ulcer in and about the stomach probably very rarely offers a hospitable shelter for bacterial growth on account of the hydro-chloric acid. In a study of anæmias Schmidt [4] called attention to the frequent association of symptoms of intestinal inflammation and indigestion with achylia gastric, and suggested that it was possibly due to bacteria let through into the bowel living, not having been killed by hydrochloric acid as usually occurs. The achylia might be a factor in the establishment of a chronic infective focus. Further down in the ileum and colon small ulcerations of any sort and perhaps more particularly undermined ulcers have occasionally been suspected as sites of infection. Goldthwaite has pointed out in certain individuals with marked visceroptosis the dependent loops may become much more permeable and intestinal organisms, usually harmless, such as B. coli, Streptococcus intestinalis, etc., may develop pathogenicity in such an environment. All of these conditions mentioned while theoretically possible are, it must be admitted, in all probability quite rare, and indeed problematic. No one so far as I know has shown the origin of a definite arthritis to be such a lesion. There are two adnexa of the gastro-intestinal tract which are very common sufferers from acute and later residual chronic infection. These are the gall bladder and the appendix. It seems likely that the joint, kidney and heart diseases referable to infections of these structures are relatively small in proportion to those in which head infections are to be blamed. The work of Billings, Rosenow and others has been published so recently, however, that very little clinical observation has as yet been recorded. A somewhat careful review of current literature has failed to bring to light reports showing gall bladder or appendix responsible for any of the series of troubles we have now under consideration.

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4 Schmidt:—Harvey Lecture, 1913.

HIGH CALORY FEEDING IN CASES OF TYPHOID FEVER IN CHILDREN.*

By H. H. YERINGTON, M. D., San Francisco.

The question of diet in typhoid fever is without a doubt the most important phase in its treatment, and although the text books during the past hundred years and long before that have devoted pages to drugs, hydrotherapy, bleeding, ventilation, etc., only a few lines deal with dietetics.

In taking up the subject of caloric feeding I thought it might be of interest to quote from a few text books on medicine written by prominent physicians during the past century.

William Cullen: Practice of Medicine, 1816: under the treatment of fevers, considered any patient with continued fever, with muttering delirium, wasting and failing heart, in the "typhoid state" and wrote as follows: "From the commencement to the close of the complaint the drinks should be diluting, mucilaginous and mild. In the latter stages, where fever and inflammation have disappeared, and debility is considerable, a small quantity of wine may be advantageously allowed. So may broths and animal jellies, but until considerable progress shall have been made in convalescence solid animal food is totally inadmissible."

At this time no differentiation was made between typhus and typhoid, the latter being only a state in continued fevers.

John Eberle, 1830: Advocated bleeding followed by a cold bath and wine. He devotes twenty pages to the treatment of typhus, one-fourth of a page being given to the diet, of which he says: "With regard to the dietetic management of the disease, it is scarcely necessary to state that the simplest kinds of liquid nourishment are alone admissible. Of these, the patient should be allowed as much as he can be induced to take, more especially during the sinking stage of the complaint." He quotes Dr. Stoker as saying, "that in the late epidemic in Ireland, many patients who were brought into the Dublin Hospital began to recover almost immediately on being allowed the free enjoyment of mild nutritious food."

John Mason Good: Study of Medicine: 1829, speaks of sitting his patients on a stool and pouring 2 gallons of cold water over their unshaved heads, which proved successful in some cases, the fever being cut short in a day or two from its commencement. This method, he says is too violent after the first three or four days of the attack, and thereafter the body is sponged. Speaking of diet he allows his patients animal broths and jellies in alteration with farinacia. This concluded his remarks on diet.

Barlow: Practise of Medicine: 1856, says under continued fevers: "This term typhoid fever is to be regarded as synonymous with gastro-enteritis. If it be understood that the intestinal inflammation in this last case is of a specific

* Read before the Sacramento Society for Medical Improvement, January 18, 1916.

* From the Department of Medicine (Pediatrics), Stanford University.

character, this doctrine can have no injurious results."

He prohibits all muscular exercise, confines the patient strictly to bed and darkens the room. The diet at this time, he says, should be of the most unstimulating kind; milk and water, or thin barley water with an occasional cup of tea, will generally be sufficient, as long as the febrile excitement is great.

Later he says: "Dr. Alison observes that the 'disease' frequently runs its course quite favorably in very foul or close air" but he himself notes that when the patients are taken out of some of the worst houses in London and put in the well ventilated wards of Guy's hospital the prognosis is better.

Loomis: Practise of Medicine: 1894, says milk is the most suitable food, but fruits are not to be allowed in any case. He advocates cold baths beginning at 70 or 80 for a temperature of 105, which he reduces to 60 until the temperature reaches 103.

Osler: Practise of Medicine: 1906, allows milk, eggs and water during the febrile stage, with no solid food until the fever has been normal for ten days. His dietary is given as follows: "Milk 4 ounces with 2 ounces of lime or soda water, alternating every two hours with 4 ounces of albumin water, made from the white of one or two eggs." He says: "it is possible that we give too much food. Of late years the disease has been treated by what has been called therapeutic fasting—little or no food only fasting."

This brings us up to the period of high caloric feeding in typhoid advocated during the past ten years by Coleman of New York, and followed by several physicians with good results since then.

During a service as house physician in Bellevue Hospital during the fall of 1909, I had an excellent opportunity of watching Coleman's adult patients during an epidemic, and caring for nine children between the ages of three and ten years of age, during the months of September and October of this same year. These cases were fed on a modified Coleman diet receiving from 700 to 2500 calories a day, consisting of a diet of eggs, broth, lactose, custards and junket. All these children held their weight or gained during the course of the disease, except one child who died of a broncho-pneumonia. This was the first series of children with typhoid as far as I know, fed by this method up to that time.

During the past three years children with typhoid coming into the pediatric wards of Lane Hospital have been given a modification of the Coleman diet.

Before going into the practical feeding of these cases, I would like to review with you the question of food values and food requirements in health and disease. In order to maintain the body weight a certain amount of proteids, fats and carbohydrates must be given. If a sufficient quantity of the two latter are given, the body proteids will be protected and not used up in the heat production of the body. Thus in typhoid fever by giving a diet high in fat and carbohydrates and low in proteids, the weight can be maintained and even increased during the course of the disease.

No general rules can be laid down regarding the food requirements which shall apply to all persons, the needs for nourishment depending on many factors, the size of the body and extent of body surface being the most important considerations.

The average man in health requires between thirty and sixty calories per kilogram of weight to maintain health and strength, and according to Langworthy, a man under varying conditions of work requires the following:

Man without muscular work.....2450 C
Man with light muscular work.....2700 C
Man with moderate muscular work..3400 C
Man with very hard muscular work..5500 C

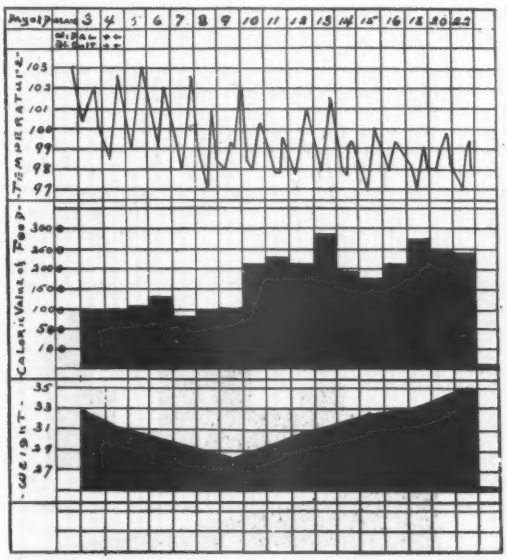

Chart C. C., 3d to 22d day. Uncomplicated case, moderately high caloric feeding. Gain in weight shown during fever stage. Dismissed on 23d day with a gain of 5 pounds in weight.

Atwater has shown that for every twenty calories developed and applied as work, eighty calories are lost in the body as heat and "internal work." Therefore in typhoid with a high fever the internal work is greatly increased, and more food than normal is needed and burned up. According to Coleman, the increase in total metabolism which occurs in typhoid fever creates a demand for more food than would be necessary to maintain the same individual in health, if he were confined to bed; the average increase during the course of the fever being about thirty-four per cent. But if the energy which the patients exert in moving about the bed is considered, ten per cent. more must be added to this, therefore five calories per kilo. must be given. If a patient's digestive organs are capable of handling this amount, the drain on his own tissues is less, his weight is maintained and his body is better able to cope with his severe bacterial infection.

If we refer back to the low caloric diets, taking

the milk and egg diet of Osler as an example, we find that his adult patients during the febrile stage were receiving less than 1000 calories per day, when they needed much more food at this time to maintain their body weight and strength.

Coleman feeds large quantities of lactate, cream and milk, giving as high as 5000 calories per day during the febrile stage.

In a large series of typhoid cases reported in 1915 by La Fetra and Schroeder they say that the loss of weight in typhoid as in starvation, and in other fevers, obeys the general law that the carbohydrate glycogen stored in the body is first utilized, and after this the fat and protein. A characteristic feature of the metabolism of prolonged high fever is the marked loss of body nitrogen. If enough food is given the nitrogen balance will be maintained and the body tissues will not suffer, thus the usual great loss in weight, lowered resistance to complications and relapses, and the toxic nervous symptoms will be eliminated.

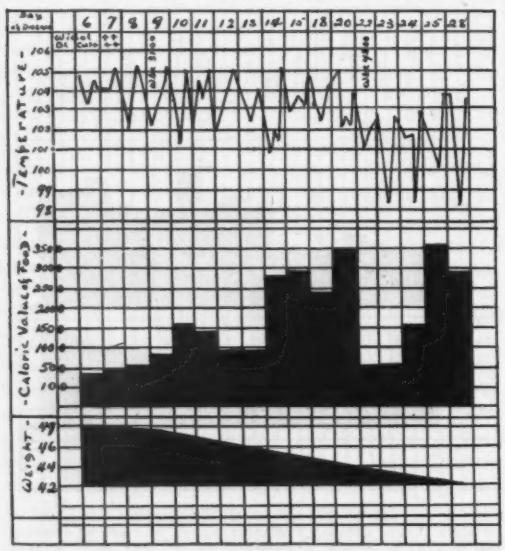

Chart of J. A., 6th to 28th day. Whey feeding 22d and 23d day for tympanites, high caloric feeding again 25th day. Disappearance of tympanites on 30th day. Dismissed on 50th day. Weight 50 pounds. Note high calory feeding during high fever stage.

The high carbohydrate diet is an ideal one for children, because large quantities of lactose can be given in most cases, especially when the fever begins to break and during convalescence.

In order to show the practical method of feeding in these cases, I will submit to you several charts, one of which, the case O. B., I will describe in detail.

This was a boy age 10, coming into the hospital in a very run-down condition in the beginning of his second week of typhoid. On admission his temperature was 104; he was delirious; white blood count 5200; Widal and blood culture positive. Up to this time he had been on a starvation diet and had lost considerable weight, his weight on admission being 50 pounds. On the fourth day the following diet was begun and given at regular intervals:

9 a. m.—Milk 4 ounces; barley water 2 ounces.... 80 C
12 m. —Milk 4 ounces; barley water 2 ounces.... 80 C
3 p. m.—Lemonade; (milk sugar 2 ounces).....240 C
6 p. m.—Milk 2 ounces; barley water 2 ounces.... 80 C
1 a. m.—Lemonade; (milk sugar 2 ounces).....240 C
5 a. m.—Milk 4 ounces; barley water 2 ounces.... 80 C
Number of calories in 24 hours.....880 C

In a few days when it was noted that the patient stood the above well, two ounces of milk in each feeding was added and another ounce of milk sugar added to each lemonade. Later the following articles were added:

Egg-nog 6 ounces (milk sugar one ounce).....270 C
Broth, clear..... 6 oz.
Ice-cream .....570 C
Custard .....270 C

The child left the hospital on the forty-fifth day after admission, getting over three thousand calories in twenty-four hours given as follows:

1 a. m.—Lemonade; (milk sugar 3 ounces).....360 C
5 a. m.—Milk 6 ounces; barley water 2 ounces...120 C
7 a. m.—Milk 6 ounces; barley water 2 ounces...120 C
9 a. m.—Custard .....270 C
12 m. —Ice-cream .....570 C
12 m. —Egg-nog .....320 C
1:30 p. m.—Milk 6 ounces; barley water 2 ounces...120 C
3 p. m.—Ice-cream .....570 C
4:30 p. m.—Milk 6 ounces; barley water 2 ounces...120 C
6 p. m.—Egg-nog; milk sugar 1 ounce.....320 C
10 p. m.—Milk 6 ounces; barley water 2 ounces...120 C

Total 3010 C

On the fourth day of admission with five hundred and twenty calories in twenty-four hours only about ten calories per pound were given. According to the history even less than this amount had been given before his admission to the hospital. By running the calories up rapidly, he was getting twenty-two plus, per pound at the end of the second week, forty at the end of the third week and sixty, calories per pound on dismissal from the hospital.

During his stay in the ward his weight had practically remained stationary with a gain of one pound on going home.

In my limited number of cases, covering possibly twenty typhoids in children, on this diet I have found the following to be true:

Two stages of the disease must be considered; first, the acute or high fever stage; secondly, the breaking temperature and convalescence stage. In the first stage in many cases, the food is not well taken, and a nitrogen balance cannot be maintained. In the second stage the calories can be increased rapidly and the normal weight and nitrogen balance maintained.

If the body is well nourished during the course of the disease, it is better able to fight the bacterial infection, and such complications as restlessness, continued high fever, tympanites, hemorrhages and perforation are prevented.

The patient is more comfortable and bright during the course of the disease, his condition is better and convalescence is shorter.

As far as the care of the patient is concerned, the time and energy given to tubbing, rubbing and applying ice coils is avoided, the nurse has a systematic way of giving food, and the food is easily prepared and the value recorded by any intelligent nurse.

I might say in closing that my object in reporting these cases is to show the practical application of a moderately high caloric diet, and compare it with other diets which I hope will be brought out by a discussion this evening.

CLINICAL OBSERVATIONS OF ONE HUNDRED CASES OF ARTIFICIAL PNEUMOTHORAX.*

By RALPH C. MATSON, M. D., Portland, Ore.

If the ever-increasing number of contributions on artificial pneumothorax may serve as a guide, it is evident that the method is constantly obtaining more adherents; but there is still a large field open to scientific study; many dark points to clear, and many difficult questions to solve, before pneumothorax therapy can celebrate its deserved triumph. While the method belongs to the most valuable in the treatment of pulmonary tuberculosis, there is no doubt that greater conservatism is being exercised, not only in the selection of cases, but in its application.

We employ the Forlanini-Saugman puncture method, using 1% novocaine as an anesthetic, introducing gas through a Soloman catheter. Since the communication of Tobieson of Copenhagen, we have used air instead of nitrogen-gas. The amount of gas and interval of repetition of injection must be individualized, using a Roentgenological, physical diagnostic and clinical control. Generally speaking, we favor small quantities of gas—200 or 300 c.c. every few days until a satisfactory collapse has been obtained, maintaining the same by using larger quantities at longer intervals. Following this plan there is undoubtedly less danger of activating a process on the other side, or getting an aspiration infection by forcing the contents of a cavity out too suddenly. By this method one also avoids the febrile reactions (not unlike a tuberculin reaction), which frequently follow large inflations. These reactions are probably due to toxins pressed out of the infected lung and swept into the blood-stream; on the other hand, it is not desirable to make more punctures than necessary, and in certain selected cases, especially for hemorrhage, the pneumothorax may be rapidly established.

During the early inflations we work with low pressures—neutral or weak positive; in fact, the lowest capable of attaining a satisfactory compression. Later, after some pleural thickening has taken place, it is necessary to work with higher pressure.

Duration of treatment should extend from six months to two or more years, except in cases of hemoptysis (tuberculosis congestiva), where treatment may be left off sooner. However, it is difficult to say when the tuberculous foci in the lung are cicatrized, even though the patient is clinically well.

Artificial pneumothorax is a method that cannot be generalized, but is limited in its employment. Not all physicians are in a position to employ it, as by incorrect application, injury can come about. One must not only understand artificial pneumothorax, which varies with the individual, but one must understand tuberculosis from a pathological, clinical and Roentgenological standpoint. Success, aside from an intelligent patient, requires a careful selection of cases, constant observation of lung changes, absolute knowledge of the size and shape of the pneumothorax cavity, and position of mediastinal contents. This information may be obtained by clinical observation and physical examination; but for every mechanical therapy of lung tuberculosis, exact, repeated, systematic observation under the x-ray with the screen and plate, is an unconditional requisite. The x-ray not only enables us to select suitable cases by establishing indications and contra-indications, but permits one to graphically follow the anatomical distribution of the lesions, their character, the degree of compression, etc., and thus safeguard the other side.

In the selection of cases the x-ray plays a very important role; in cases with extensive, destructive changes on the one side, producing marked auscultatory phenomona, it is exceedingly difficult to pass judgment upon the integrity of the opposite side, especially behind, where rales are often transmitted to the opposite side with such intensity that refined tones upon the good side cannot be heard. Even in 1856 Feuger called attention to the echo of bronchial breathing from the diseased to the healthy side. Since then, Budde and others have called attention to the conduction phenomenon of rales. At times one is astounded at the amount of disease shown upon the plate; cases that from a physical diagnostic standpoint have been considered one-sided, are frequently seen to have extensive disease upon the opposite side, especially when a bronchogenic infection of a central portion of the lung has taken place (Chart I). The x-ray reveals the character and extent of this type of infection with a degree of certainty not otherwise attainable.

Furthermore, in cases with severe acute infiltrating disease upon one side and compensatory changes upon the other, the respiratory sounds are so altered that it is difficult to determine the extent of the disease.

Hemorrhage cases are often puzzling; a small rigid wall cavity may be a source of much annoyance and continue to bleed on account of lack of compression. The x-ray clears up the difficulty; by its use one also avoids the not uncommon mistake of trying to compress a supposed cavity, which proves upon the plate to be a deviated trachea.

From a Roentgenological standpoint, circumscribed hilus tuberculosis, characterized by round and oval uniform spots; hilus-apex tuberculosis with isolated areas in the apex; increased peribronchial or perivascular paths; also somewhat more extensive disease with scattered areas in apex and upper lobe but isolated in lower lobe, do not contra-indicate the compression of the other side. However, if we find in the upper lobe confluent patches amounting to consolidation, broncho-pneumonic or destructive processes, compression is contra-indicated.

Needless to say, the x-ray findings must be interpreted with great caution and only by one thoroughly familiar with pathology of tubercu-

* Read before the annual meeting of the California State Medical Society and the California Association for the Study and Prevention of Tuberculosis, Fresno, Cal., Apr. 19, 1916.

losis. The interpretation must be done by the clinician himself. One cannot differentiate active from inactive tuberculosis by means of the x-ray alone. Great caution must be exercised in diagnosing cavities from the plate alone. Bands of adhesions circularly arranged so frequently resemble cavities that even an expert will make mistakes if his work is not controlled by the physical examination and clinical history.

The indications for an artificial pneumothorax vary within the widest limits, and every individual case represents a problem for the physician to solve on the basis of his experience in the usual and pneumothorax therapy. Early infiltration cases should be withheld for the present as they usually recover by other methods; and in some cases the lung shows little tendency to expand after prolonged compression, on account of the tremendously thickened pleura. Furthermore, compression does not absolutely prevent extension of the disease into the compressed part. Therefore, in a given case, if an early one, we must consider the probability of recovery by the usual therapy; or whether it is better to possibly sacrifice the uninvolved lobe on the affected side to the method and save the other side, and perhaps the individual's life; especially if the case is severe enough, and the result of the usual therapy, after a reasonable trial, is in doubt.

The best results will be achieved in the chronic forms, preferably during the period of infiltration, with destructive lesions scarcely initiated (or at least, just installed), adhesion-free and positively unilateral. This cannot be too often emphasized. One sees splendid results in cavity cases if the cavities are not extensive or superficial; in the latter case not only is there great danger of rupture of lung tissue under compression, but adhesions are frequently present over superficial cavities, preventing a satisfactory collapse. In cavity cases, the sooner an artificial pneumothorax is instituted, the better. Not only is the danger of intestinal infection diminished, but the possibility of bronchogenic extension on the same, and to the opposite, side is lessened. To use the method when compelled to, invites disaster; the danger of delay far exceeds those associated with the procedure or damage to the lung. We believe that Forlanini is correct in advocating the method when excavation begins.

In severe hemoptysis, artificial pneumothorax is strictly obligatory; not only does it put a quick end to bleeding in the absence of adhesions, but it lessens the danger of aspiration pneumonia or aspiration tuberculosis.

That pneumothorax cannot do harm if it does no good, is certainly not in accordance with our experience. Cases with an old extensive, fibrocaseous disease on one side and acute infiltration on the other are at times misleading; acute infiltration or inflammatory processes on the noncompressed side of bilateral cases are very likely to be made worse. Not too severe changes (probably limited to infiltration) in the good side, seem to be favorably influenced by pneumothorax; but, according to our experience, the more severe are only occasionally lastingly improved; lesions limited to the apex or extending from the hilus outward appear most favorably influenced. Lesions of the base of the uncompressed side are likely to be made worse; it is in this class of cases that the x-ray again renders such splendid service. In bilateral cases one obtains prolonged relief of symptoms—rarely cures; the end results are always doubtful. If bilateral cases are contemplated it is foolish to wait for the patient to get in better condition; possibility of benefit from compression may be lost. Whether a case of this character will be improved or harmed cannot be foretold. But, if bilateral cases are done, the disease on the good side must be limited to the upper lobe and must not be acute; it must be carefully watched and the collapse cautiously brought about.

As a matter of fact, one should not aim to collapse the lung in this class of cases; a layer of gas between the visceral and parietal pleura is sufficient to interrupt the circulation of lymph, causing local toxin absorption.

Actively advancing and caseous-pneumonic forms appear unsuited; in the latter, for anatomic reasons, no effectual collapse can be hoped for. Forlanini warns us against it in these forms and reminds us that it is frequently bilateral and should not be used except as an extreme measure, and then very cautiously. Bilateral cases with softening on the less affected side, or infiltration in the lower lobe without softening, are likely to be made worse. Isolated lower-lobe tuberculosis, on account of the accompanying bronchiectasis and difficulty of collapse, appear not adapted. Cases with a rapid course and severe toxemia do not do well; not only theoretically can little be expected, but in practice this is confirmed. Long standing cases of fibroid tuberculosis with cavity formation are not so satisfactory as one would be led to believe; not because of the method, however, but on account of the likelihood of adhesions which prevent satisfactory collapse. This type of tuberculosis represents the most difficult cases to obtain a clinically effective degree of compression. Success cannot be expected without resort to appropriate surgical procedures, which, as yet, have not had wide application. Forlanini and Fagioli have reported success in desolate bilateral cases compressing both sides. We have had occasion to resort to this in four cases, with one complete success.

Unfortunately, many cases exhibiting indications for an artificial pneumothorax cannot be compressed on account of pleural adhesions; it would be splendid to have a method that would enable us to recognize free pleural space from one partially or wholly obliterated with adhesions, but there is no absolute criterion that permits a differentiation in every case, between the normal and pathological pleura; the value of the x-ray for this purpose has been exaggerated; the excursion of the diaphragm even under fluoroscopic screen permits no conclusions; for in cases of diffuse lung changes with involvement of the lung-border, the

phrenico-costal sinus may appear obliterated and lung excursion fail through border adhesions, yet offer no real obstacle to gas entry. On the other hand, in cases with high lateral adhesions, little interference with diaphragm movement or lung-border excursion takes places. X-ray plates may be clear, yet collapse found impossible.

Physical examination undoubtedly gives more reliable information than the x-ray regarding the presence or absence of adhesions. Generally speaking, the greater the mobility of the lung-border, the greater the probability of free pleura. Important hints of existing adhesions are marked weakening of the breathing over the lower lobe; local shortening of the inspiration ,Jacquerod's sign); presence of the "dissociation symptom" of Kuthy—that is, where the pectoral fermitus is weakened with increased bronchophony over the same dulled area; and the demonstration of diminished lung excursion by percussion; however, no case should be selected upon the data gathered from the physical examination alone, for one is occasionally agreeably surprised in establishing a perfect pneumothorax in a clinically adherent case.

For an intimate knowledge of the mechanical condition occurring in the thorax after having introduced gas, we find clinical physical diagnostic methods of little service in determining the size and character of the pneumothorax. Especially is this true of the small, flat variety, since neither tympany nor weak breathing can be demonstrated. With a large pneumothorax and strongly compressed lung, the breathing is distinctly weak or absent; rales are very faint and distant, if at all present. In case adhesions prevent certain lung portions being entirely cut off from breathing, one hears through the gas distinct breathing of a peculiar, ringing quality, similar to amphoric breathing. Rales, if present, have a characteristic metallic sound. By percussion one obtains in total and large pneumothorax, tympany over the whole thorax; also often over the non-compressed side, due to the compensatory changes; so that one can be in doubt as to which side has been compressed. Of course, here the breathing shows clearly; one hears over the non-compressed lung, intense, sharpened vesicular breathing. Physical signs may indicate complete compression, but the x-ray will show many cases uncompressed. So many interesting and important phenomena are shown by the x-ray that are not capable of recognition by any other method, that it is an absolute necessity.

Rubino has pointed out the importance of noting the diaphragm tonus. This muscle suffers in the wasting process in tuberculosis, as do other muscles, particularly the chest muscles; in women especially can this be observed. Diminished diaphragm tonus permits the diaphragm to be pushed downward as gas is introduced, increasing the intra-abdominal pressure. Many patients, especially in the beginning of treatment, complain of fullness of the stomach, loss of appetite due to downward pressure upon the stomach in left-sided cases; in other cases one observes nausea from downward pressure upon the liver; also loss in weight, insomnia and extreme nervousness. This mal-position of the diaphragm is only recognizable radioscopically. Brauer designates this symptom complex "vagus dyspepsia" and attributes it to vagus pressure. The condition is relieved by reducing the intrathoracic pressure.

The position of the heart and mediastinum is important. In general we observe that in cases free of pleural adhesions, the mediastinum bulges only after the lung is fully compressed; but at times one observes great mediastinal displacement after comparatively slight intra-pulmonary pressure, especially if adhesions are present. At times this displacement appears so great as to make one very anxious; yet the patient complains of little. Nitsch in the Brauer clinic, upon the basis of anatomical studies, has pointed out two weak places in the mediastinum; first, in the anterior mediastinum between the second and fourth rib; second, in the posterior mediastinum in its lower part, between the spine and aorta behind, and the oesophagus in front. The former, the weaker of the two, often forms an actual mediastinal hernia. These mediastinal hernia are clearly seen as half-moon-shaped gas areas, bulging toward the uncompressed side; too great pressure not only may defeat the object of compression by bringing too much pressure upon the other lung, but rupture at Nitsch's point may occur. The manometer is not a reliable guide to avoid these accidents, since low-pressure readings may occur with weak mediastinal pleura. The x-ray shows clearly the position, not only of the mediastinum, but also the trachea, heart and blood-vessels. In the absence of an x-ray it is a mistake to compress until no rales are heard, for one often hears large, metallic rales, due to the movement of mucous in the large bronchi taking on metallic quality by resonance in the pneumothorax cavity.

While the x-ray is of such great value in the selection of cases and in familiarizing us with the size and character of the pneumothorax, we must rely chiefly upon the stethoscope to detect early damage to the opposite side. A careful watch and record of the good side must be kept. After the first few inflations, when the diseased lung has been placed somewhat at rest, one notices very often a marked reduction of rales in the good lung as a consequence of a diminution of the resonance phenomena. One should orientate himself regarding the character, quantity and extensiveness of rales on the good side, if present, and their topographic location. Weekly examinations and a careful record will tell whether they increase. In many favorably progressing cases, as a matter of fact, they become less. In case they appear over a wide area, one must either diminish the compression or leave off altogether.

The benefit derived from a pneumothorax in our cases has been almost in direct proportion to the size of the pneumothorax. In chiefly unilateral cases free of adhesions, and which are otherwise suitable, one obtains arrest and healing of the pathological process. When the mechanical action of the gas is prevented through adhesions and in bilateral cases, one may obtain symptomatic results.

Adhesions between lung and diaphragm are the

most harmless. Apical adhesions are serious; but the most serious were those attached to the lateral aspects of the chest-wall. In selected cases the energetic use of gas under pressure will separate recent adhesions; 20 to 30 c.c. H_2O should be the average limit, although a much higher pressure may be used in some cases. We have used as high as —80 H_2O with success in two cases, where we re-established a pneumothorax that was becoming obliterated by secondary pleural thickening. However, high pressures with a complete pneumothorax are dangerous; cases must be carefully studied with the x-ray before resorting to high pressures, avoiding, if possible, old and extensive tuberculosis where cavities and caseous areas in the cortex are always present, representing the source of one of the most frequent and serious complications of the treatment; namely, lung-perforation. Gas embolism, hydrothorax and pleural eclampsia are also dangers to be considered in forcibly separating adhesions. Adhesions should be given plenty of time to thin out; one must not attempt rapid stretching; if they are band-like, the Jacobeus endopleural operation with cautery through a thoracoscope would seem to be an ideal procedure.

The operator must be thoroughly informed, not only in the indications but in the accidents and the means of preventing them. Since the experiments of von Muralts on intra-thoracic pressure values, the observation of the manometer has become of greatest importance, not only to safeguard the heart and other lung, but the life of the individual. Let it be an absolute rule not to introduce gas before you are convinced the needle is in free pleural space, or before a well-defined manometer reading swinging more than 1 c.c. H_2O and registering on primary punctures' negative pressures. Failure to observe this rule may give one a painful experience, never to be forgotten, and endanger the patient's life. A merely swinging manometer does not indicate free pleural space, and if still in doubt, it is advisable to use the "Holmgren procedure." The manometer tells the position of the needle; if it has entered a bronchus or cavity (air containing lung tissue), and superficial quiet breathing occurs with open glottis, the manometer shows an excursion above and below the neutral point, usually a +1 —1. By deep breathing the variations may be 2 c.c., but never beyond. Furthermore, one will observe (the patient breathing slowly) that the —2 occurring during inspiration, falls to neutral before the expiration begins; and the +2 exists only with the beginning of expiration and falls to neutral before the inspiration begins. If the patient holds the breath with glottis open, either at inspiration or expiration, the + or — sinks to neutral. The explanation, of course, is simple, for pressure variations in air containing lung tissue when the glottis is open, exist only during respiratory movement. Furthermore, should gas be admitted, it enters with suspicious rapidity and the patient feels no signs of pressure.

Negative pressure readings with slightly moving manometer are also present in case the needle enters a cavity with obstructed opening. Gas entry can cause gas-embolism by tearing a small vein. Should the needle enter a lung vein, it is said the manometer registers a negative pressure, which is increased by deep breathing. In case a pulmonary artery is entered, the pressure is — and almost stationary, but cardiac readings will be noted. If the needle be in solid tissue, the manometer registers neutral and no movement takes place. If the needle be in the pleural cavity, the manometer at once shows two characteristics: first, movement essentially greater than above mentioned; second, it maintains itself at the end of the respiratory phase and does not sink back to neutral, the pressure being invariably negative in primary punctures,—5 to 10 c.c.; generally speaking, the larger the thorax, the greater the negative pressure. A reverse curve—one that is + on inspiration and — on expiration—occurs in pneumothorax only in "paradox diaphragm movement"; however, the latter does not always produce reverse pressure values. The "paradox diaphragm movement" can occur from too great pressure in the pleural cavity.

The manometer also gives accurate information regarding the presence or absence of adhesions. In case the pressure gradually increases as gas is let in, and no pain complained of, it speaks for the absence of adhesions. On the other hand, if the pressure becomes high after the introduction of relatively small quantities of gas, it indicates adhesions and the patient complains of much pain, often referred to the shoulder or stomach—more frequently to the former.

Complications. Aside from an occasional mild subcutaneous emphysema, the most frequent complication was hydrothorax; this occurred eight times. Generally speaking, it has been our policy to let it alone, provided it caused the patient no inconvenience and maintained compression. Frequent aspirations should be avoided. It is not within the scope of this paper to discuss the etiology of hydrothorax complicating pneumothorax; however, our effusion cases divide themselves into two groups; group one comprising four cases where, on account of adhesions, it was necessary to work with somewhat high pressures, and tearing adhesions could easily be ascribed as a cause; in one case the fluid appeared after the sixth month, ushered in with fever; it remained two months and absorbed, during the patient's absence from supervision, leaving the lung partially adherent and tremendously thickened. A pressure of 80 c.c. H_2O was necessary to re-establish the pneumothorax. Marked retractive processes, followed by diffuse pleural thickening and diminution of the pneumothorax cavity as a consequence of connective tissue proliferation, occurred in this case. The cavity has been dry now for two years. The other three cases of this group occurred early, accompanied by fever at onset, and were treated by aspiration; in one the fluid has disappeared; in the other two it is still present. Group two comprises four cases, wherein the effusion was probably due to the extension of a cortical tuberculosis through the pleura. In one case it appeared after the third month, without fever. The patient was compelled to remove to a distant place where competent medical super-

CHART I.

GENERAL SUMMARY OF ALL CASES.

CLASS OF CASES		No. of Cases	Bilateral	NUMBER with COMPLICATIONS	Satisfactory Collapse	Partial Collapse	No free pleural space	RESULTS OF TREATMENT: Clinically well	Arrested Ambulant	Favorably influenced	Temporarily Improved—Abandoned	Unimproved	Made worse	Still under	Dead	COMPLICATIONS UNDER TREATMENT: Gas Embolism	Pleural shock	Sterile Exudate	Sterile Empyema	Empyema T. B. demonstrated	Empyema due to pus bacteria	Lung perforation	Hemoptysis compressed side	Extension of disease other side	Secondary pleural adhesions	Pleurisy with effusion opposite side.
Severe acute infiltrating and caseous pneumonic forms		20	15	2 larynx 1 bowel	15	4	1	1	3	3	2	8		9	7	0	0	1	1	1		1				2
Chiefly unilateral, destructive, fibrocaseous, extensive and progressive		19	2	3 bowel 1 larynx	13	5	1	6	4		6	2		6	4	0	1	5	1			1	2		2	
Chiefly unilateral, fibrocaseous, cavernous, cavity manifestation in foreground		16	1	1 bowel	3	5	8	1		2	3	10		3	4	1										
Severe and repeated Hemoptysis		6	2	1 sarcoma of testicle	4	2	0	1	1	2				1	3											
Tuberculous Pleurisy with effusion		8	0	1 tuberculous meningitis	7	1	0	7							1											
Non-tuberculous lung disease	1 post pneumonic empyema				1			1																		
	2 abscess				1		1	1				1														
	3 bronchiectasis				1	1	1			1		3					1				1					
	1 gangrene				1						1				1											
	3 diagnostic				3																					
	Total	10													3											
Bilateral Fibrocaseous cavernous		12	12	2 larynx 4 bowel	10	2	0	0	1	1	6	1	2	2	8			2				1		2		
Ultimatum refugium Desolate Bilateral Cases.		9	9	4 larynx 3 bowel	7	1	1	0	0	0	3	5	1	0	8									1		
TOTAL and %		100	41	11 bowel 9 larynx	66	21	13	18	9	9	21	29	3	21	39	1	2	8	2	1	1	3	2	3	2	2

vision was not available; his improvement was constant; he returned for examination two and a half years later, believing himself well, as he had no subjective signs of tuberculosis; he complained only of dyspnea on exertion, which he attributed to adhesions, as he had not felt fluid for more than one and a half years, and presumed it had absorbed and the lung re-expanded. Examination showed the heart to the right of the sternum and the left pleural cavity filled with a sterile, chylous exudate containing debris, fat and cholesterin crystals; the pleura was not markedly thickened. The fluid has been withdrawn and the lung has re-expanded.

A second case of this group formed fluid after the fourth month; the patient's general condition was made worse. Two cases became purulent and later got a lung perforation.

Pyothorax occurred seven times; three were sterile, two causing neither fever nor constitutional disturbance; one was due to infection with pus bacteria; all were treated with aspiration with gas insufflation.

Three were due to lung perforation. One was referred to the surgeon and died while a rib resection was being done. Two were drained under local anesthesia; one got an arrestment and is ambulant with a tube in place, more than one year. The other case succumbed to an aspiration tuberculosis on the opposite side.

Pyothorax in our cases, except one (pyothorax occurring in a bronchiectasis case after an acute tracheo-bronchitis), was probably due to the perforation of some cortical caseous focus. Aspiration followed by irrigation of the pleural cavity with 1-5000 potassium permanganate solution or .5 Na, Cl. Sol. and gas insufflation has proven effectual; hence greatest care must be exercised not to work with high pressures on account of the danger of lung perforation, which has a gloomy outlook. After pleural thickening has taken place, little febrile disturbance is seen, due, no doubt, to the absence of toxin absorption. A very unpleasant complication, or what might more properly be designated sequela, which frequently renders further pneumothorax treatment impossible, consists in the formation of secondary pleura thickening and adhesions. We have had two cases in which the pneumothorax cavity became gradually obliterated in spite of high pressures and frequent inflations. The cause is no doubt due to the irritation of the pleura.

Air embolism occurred once early in our work; the patient was unconscious 25 minutes with a hemiplegia; full recovery took place within one hour.

Pleural eclampsia occurred twice, in both instances on reinflations of nervous, apprehensive women (one six months pregnant); both were adhesion cases and in both instances it came on after the needle had been withdrawn—one immediately after and the other ten minutes after. Both cases were characterized by the typical symptom complex. Psychic symptoms, numbness, loss of consciousness, mental confusion, tonic contractions on the pneumothorax side, no loss of bladder or bowel control, no vomiting, rapid pulse, weak and irregular; pale skin with cyanotic spots, especially on face, throat and chest; breathing superficial and irregular. The attacks lasted one-half hour. Recovery without complications took place in both instances; one case (after a preliminary morphin-atropin injection) has since been reinflated several times without event, using warmed nitrogen gas.

Recrudescence in the uncollapsed side that could be justly attributed to the pneumothorax, occurred three times; in two cases withdrawal of the gas caused its subsidence; the other was progressive.

Pleurisy with effusion on the opposite side has occurred three times; two were bilateral cases with a moderately active lung lesion on both sides, complicating a pleurisy with effusion; aspiration of fluid and replacing it with gas brought immediate improvement; and while under compression, the opposite side became active with exudate formation, which was also aspirated and replaced with gas, establishing a double pneumothorax. One case was progressive; autopsy showed tuberculous laryngitis, tuberculous pericarditis and tuberculous enteritis; there was no active lung tuberculosis. The other case was under treatment with a double pneumothorax one year; both sides have since re-expanded; the patient is arrested and able to work.

Critical study of our cases reveals that failures to induce a clinical cure have been due to the independent evolution of pre-existing lesions in the other lung or elsewhere, or to pleural adhesions, preventing a satisfactory collapse.

AIMS OF NATIONAL FOOD ADMINISTRATION.

The primary aim is to see that the people of this country eat a sufficient quantity of food, but not an excess, and that they stop waste. It is also to reduce the consumption of staples so that a large amount may be set free for export to the allies.

We wish to urge in particular the free use of vegetables and perishable foods where they are produced, to encourage the preservation of perishable and semi-perishable fruits, vegetables and other foods, to substitute other cereals to a large extent for wheat, and to reduce materially the consumption of meat.

Many other phases of the work will be developed from time to time, and reported regularly to the councils of defense.—Herbert Hoover.

We, the public officers of this country, must overcome the inertia of this habit in the community and offset the momentum of great industries by teaching the consumer and the producer of alcoholic beverages to discontinue their mutual conspiracy, which is robbing the future generations of their birthright of health.—Haven Emerson, M. D., Amer. Jour. Pub. Health, June, 1917.

TUBERCULOSIS IN CHILDHOOD WITH UNUSUAL MANIFESTATIONS.

By LANGLEY PORTER, M. D., San Francisco.

The subject assigned to the speaker, tuberculosis in childhood, is obviously too extensive to be discussed in twenty minutes. It would be hopeless even to attempt a review of the literature on any subdivision of this subject, for volumes have been written on the etiology, the symptomatology, the prophylaxis, the treatment and a hundred and one of the important side issues that appeal to those who make a study of the subject.

In going over the last eight years' records of children who have been treated for tuberculosis at the Stanford University Medical Clinic, I have encountered a number of histories which record unusual findings and it seemed to me that the section might be interested in the report of the more remarkable of these. I have therefore determined to present four of these histories in some detail.

The first of these and one of the most unusual cases of tuberculosis of the peritoneum we have encountered, was that of A. B., age 13 months. This child was admitted to the children's ward of the Lane Hospital on February 28th, 1916. He was a well grown, emaciated child of 13 months, weight 25 lbs. He had only four teeth. The physical examination, except for the abdomen, was negative. It is recorded that the abdomen was rigid, somewhat distended, tender to palpation and rigid, so that nothing could be made out by palpation without an anesthetic. Under an anesthetic there was felt a curious crepitation which seemed to rise from the small intestine that had been pushed forward and was supported by an indistinct mass or masses in the abdominal cavity. The liver and spleen seemed normal. There was no evidence of inflammation in the right iliac fossa. The right testicle was indurated, hard and apparently not tender. The right spermatic cord was in the same condition, about as large as a goose quill. The child lay with the knees bent and the thighs flexed on the body; extension of the legs apparently gave pain.

On examination under an anesthetic, the child vomited at least 1½ pints of fluid, which was largely bile, with some curdled milk. The stomach was washed and a further large quantity of bile stained fluid was withdrawn.

The family history was unimportant. The personal history revealed nothing except that for a long time the child had been unwilling to lie on its back with its legs extended. He had been perfectly well on January 22nd, while on the 23rd, on waking, he had an attack, which, as described by his parents, must have been a chill which was followed by cyanosis and coldness of the extremities for about three hours before fever supervened. The child showed an increase of white blood corpuscles to the number of 18,400 and 63% of these were polymorphonuclear. The urine showed a moderate number of finely granular and hyalin casts. The stool examination gave no evidence of any disturbance in the digestive tract. Lumbar puncture was done; there was no apparent increase of pressure, although this was not measured. Total cells in the fluid were 11 per ccm. Nonne and Noguchi tests were negative. Sugar reduction was positive. X-ray examination showed the abdomen distended with gas; the bismuth began to leave the stomach in one hour; and the end of 24 hours there was considerable bismuth still present in the stomach. The remainder was irregularly scattered throughout the bowel which showed a marked delay in the emptying of the stomach and irregular passage of bismuth through the bowel. The child died on March 6th.

Autopsy showed a very well developed, much emaciated male child of about a year of age. Pupils slightly dilated, equal. Skin and visible mucous membranes were pale. No enlargement of superficial lymph glands. Abdomen was markedly distended, slight edema of feet and lower legs. The right testicle was about four times normal size, freely movable in the scrotum. Marked congestion of the back—almost a complete atrophy of subcutaneous fatty tissues—muscle was poorly developed. The peritoneal cavity was entirely obliterated by easily broken adhesions. The small intestines were covered with a layer of fibrous exudate which completely enclosed them. Loops of the small bowel were bound together by fairly firm fibrous adhesions. On the fibrinous exudate between the folds of the mesentery were many fine grayish nodules. The appendix was rather short, bound down in the mass which surrounded the caecum, otherwise normal. The mesenteric lymph nodes were enlarged; largest 2 cm. in diameter, filled with many just visible grayish white nodules. The retroperitoneal lymph nodes were apparently not enlarged, stomach bound down to diaphragm by adhesions which also surrounded the liver and spleen. The spleen was of normal size, external surface covered with a layer of fibrin, cur surface showed a fairly firm pulp, apparently normal. The right spermatic cord was thickened. The testicle was normal; in upper part of epididymis were several just visible grayish nodules. Similar grayish nodules were seen along the course of the spermatic cord. The left adrenal was normal. The right kidney was somewhat swollen, external surface was smooth, cut surface showed some widening of the cortex, markings were apparently normal. The left kidney was the same as the right, somewhat paler. The bladder and rectum were normal. The duodenum contained bile-stained contents. The bile duct was patent. The stomach was not dilated; it contained bile-stained mucus. Mucous membrane apparently normal. The abdominal aorta was normal. The liver was covered with a layer of yellowish fibrin. The cut surface was of a bright yellow color; centers of lobules stood out as reddish areas. The right lung showed a diffuse emphysema in anterior, middle and lower lobe. In the lower lobe there were large areas of collapse. Scattered throughout the lungs and on pleural surface were many grayish nodules with an oval hemorrhagic area around them. Peribronchial glands normal. The left lung same as right. Nodules may be squeezed out and were quite discrete. The heart was of normal size;

valves and muscle normal. The thymus was not enlarged.

Diagnosis: Tuberculosis of the peritoneum. Tuberculosis of the mesenteric lymph glands.

Smears of peritoneum stained with Gram method showed many lymphocytes and large endothelial cells, few polymorphs, no bacteria. Smears of peritoneum stained with acid fuchsin showed many acid fast granular bacilli, the greater part of which were contained in the cells.

Smears of the lung: Gram method showed polymorphs in moderate number, lymphocytes and a moderate number of Gram positive diplococci. Acid fuchsin stain showed a large number of acid fast granular bacilli.

Peritoneum: Marked cellular thickening in which there were numerous large caseous areas surrounded by epithelial cells. Few small cellular nodules.

Lung: Few recent tubercles.

Heart muscle: Normal.

Liver: Extreme fatty infiltration, many small cellular tubercles.

Peritoneum: Covered with partly organized fibrin. Some tubercles in granulation tissue.

Stomach: Normal, except for tuberculosis of peritoneum.

Lymph gland: Marked catarrh in lymph spaces. Several recent tuberculous areas and beginning caseation.

Spleen: Marked passive congestion. A few caseous spots. Capsule showed adhesions in which were numerous caseous areas and tubercles.

Kidneys: Normal.

This case is of interest chiefly because it illustrates how extremely acute a case of tuberculosis of the peritoneum may be and how difficult the diagnosis may be made. The rigidity of the abdomen led to the belief that there were deep masses of retroperitoneal sarcoma present. The acute onset and rapid course coupled with extreme infiltration of the testicle and cord further confused the diagnosis. The character of the urinary findings was unexpected in a case of tuberculosis of the peritoneum and the blood picture was such as to force the consideration of some other infection than tuberculosis.

Another odd and unusual case was that of J. T., who was sent to the Lane Hospital by Dr. C. S. Rodgers, of Watsonville, Calif. The boy was suffering from extreme enlargement of the glands of the neck. To such a degree were these glands enlarged, that the superficial clinical picture was that of Hodgkin's disease. Summary of his physical examination, made at the time of his admission, states that he was a well nourished Italian boy, 12 years of age, whose physical abnormalities consisted only of marked enlargement of the preauricular anterior and posterior cervical glands and lesser enlargements of the axillary and inguinal glands. He had some opacities of the cornea and was unable to speak above a whisper. The diagnosis of Hodgkin's disease and interstitial keratitis was made. His chest revealed no evidence of tubercular involvement. In the blood picture the red cells were entirely normal, both quantitatively and qualitatively. W. B. C. 8350, Polys. 66%, lymphocites 26%, large monos and esinophiles each 3%. A gland was removed from the posterior cervical triangle. This was about the size and shape of a large almond, pinkish gray in color, fairly soft in consistency. It cut easily and cut surface was smooth and gray in color with numerous small yellowish areas scattered throughout, but no areas of definite caseation. Under the microscope there was diffuse proliferation of lymphoid and endothelial cells. The lymphoid follicles had entirely disappeared. Scattered throughout the sections were many well defined tubercles with giant cells. Some of these showed beginning caseation. There was a moderate increase in the connective tissue of the stroma.

Following this report an extensive dissecting operation was done and about 50 glands were removed; these varied in size from a hen's egg to a small marble. The report of the pathological findings duplicated those just read and it was obvious that the child was not suffering from Hodgkin's disease, but from a tuberculous lymphangitis.

A more characteristic case of tubercular peritonitis is that of K. L., age 3 years. Complaint on entrance was constipation, vomiting and abdominal pain and flatulence. Family history was unimportant. Patient's previous history was one of difficulty in feeding, although she weighed 26 lbs. at 1 year and although she erupted her teeth, walked and talked at the normal time. She had mumps and chicken pox. Nine months before her entrance it was noticed she was severely constipated and had marked abdominal distention, which gradually became worse. For two months constipation had been extreme, she had vomited very frequently and complaint of pain in the belly. Pain was sometimes complained of just before urination. On some days vomiting was so extreme that no food was retained. The blood picture was W. B. C. 6150, of which 58% were polymorphonuclear. Wassermann was negative on two occasions. Nothing abnormal in the urine, and the stool showed that both fats and ordinary starches were well digested. An X-ray taken after bismuth enema discovered what was apparently an annular constriction in the sigmoid. A plate taken after a bismuth meal apparently confirmed this finding. Dr. Stillman operated on the child. making a median incision in the lower part of the interval between the rectus muscles. This revealed: Peritoneum adhered to mass underneath and by easily broken adhesions to underlying intestines. A large solid caseous mass was found in the parietal peritoneum extending across the abdomen. The upper edge at the level of the umbilicus—hard and sharp. This mass was about two inches wide and half an inch deep. The visceral peritoneum was red, sticky and covered with small tubercles, the loops of the intestine all being matted together.

The retroperitoneal and mesenteric lymph nodes were all large and hard. There was a large hard mass in the region of the sigmoid.

For some time after the operation she seemed to be relieved and a month later was allowed to go home, but immediately returned to the hospital because of vomiting and constipation with very marked distention of the upper bowel. This time a reversed peristaltic wave was seen over the upper abdomen, which disappeared after vomiting. She did not gain weight, but looked pretty well, then became apathetic and the symptoms continued. The child went progressively from bad to worse. An obstinate constipation alternate with diarrhoea then set in. Then almost complete obstruction of the bowel supervened and when the child seemed to be on the point of death, she developed a purpura and a fecal fistula. From this time on her condition improved rapidly and she was dismissed from the hospital three months after the first operation in apparently good and improving physical condition. Since her dismissal she has continued to improve and gain strength and weight. I am indebted to Dr. Herbert Yerington for a report of this case.

In a large proportion of our very young babies who have succumbed to tuberculosis, the initial lesion is that of the middle ear.

In eight years, we have had some 20 children under 2 years die of tuberculous infection; the following is the youngest among them:

B. B., a male child, age 2½ months, born at full term on January 17, 1913, never seemed to thrive. Wassermann was negative. On the left side there was a facial paralysis and also swollen glands on that side of the neck. The child died on March 31st, when 2½ months of age. Diagnosis of tuberculosis of the middle ear had been made. Autopsy revealed a very poorly nourished infant, 38½ cm. long, in very much emaciated condition. The legs showed a slight edema, the skin and visible mucous membranes were pale. There was a marked enlargement of the lymph glands on the left side of the neck, also marked enlargement on the right side. The subcutaneous fatty tissue showed complete atrophy and muscles were poorly developed. The position of the abdominal viscera was normal; thymus gland was quite small, measuring only 2½ x 2 x ½ cm. The heart was small, but otherwise normal. There were no tuberculous glands in the posterior mediastinum on the left side, although the left visceral pleura and left lung were found to be studded with innumerable small grayish white tubercles. The bronchial tubes were normal but a few apparently recent tubercles were found in the peribronchial lymph nodes. In the anterior part of the upper lobe, the tubercles are somewhat larger. The center of one of the groups contained a small hemorrhagic focus, 3 x 2 mm. The lung, bronchi and peribronchial lymph nodes on the right side were similar to those on the left. At the right apex there were two somewhat larger areas of red consolidation with numerous tubercles about 5 mm. in diameter. The spleen was enlarged, 5½ x 3 x 2 cm. and contained several large tubercles, the largest about 3 mm. in diameter, numerous small ones also being present. Both kidneys were small and showed some cyanotic spots. The cortices contained a number of small tubercles. There were no enlarged glands in the retroperitoneal region. The mesenteric lymph glands showed slight enlargement, but no visible tuberculosis. One of the Peyer's patches showed a yellow spot surrounded by a hyperaemic area. A lymph node with a large area of caseation was found at the hilus of the liver, which was hyperaemic, swollen and studded with innumerable small gray nodules. No lesion was visible in the tonsils, pharynx, trachea or tongue. On the left side of the neck there was a large mass of tuberculous glands, 4½ x 2 cm., the center of which contained a large amount of broken down, cheesy material; on the right side some glands of moderate size with small nodules. The right lobe of the thyroid also contained tuberculous nodules. Brain was found normal, also the hypophysis. The tuberculous glands on the left side of the neck extended up to the base of the skull, where a large caseous mass began, which extended into another focus in the pyramidal portion of the left temporal bone. Caseation surrounded the middle ear on all sides. Microscopically tubercle bacilli were found in lung, liver, spleen, kidneys, lymph glands of the peribronchial region and mesentery, the neck and the caseous masses of the middle ear.

This case was quite evidently one of the rarely occurring cases of transplacental tubercular infection, sometimes called congenital. Pehù and Challier, who have searched the literature for reports of such cases, were able to collect only 51. It is obvious that in most instances the infection is the result of a bacillaemia occurring after the placental circulation is well established. However, Grulee, who has recorded one such case, considers that there may be two forms of such infection, which he denominates pulmonary and abdominal, and he says that the pulmonary form is not always of placental origin and may result from the aspiration of tubercle bacilli together with birth discharges at the time of birth. In the case reported here, the oldest lesion was undoubtedly the caseous nodule at the hylus of the liver and it is undoubted that, although the pulmonary symptoms were predominant, that this was a true case of infection through the placenta.

Recent action of the U. S. Department of Commerce in recommending China to American patent medicine interests as a good field in which to develop their business, is unanimously condemned by anti-tuberculosis workers all over the country through a resolution adopted by the National Association for the Study and Prevention of Tuberculosis made public to-day.

It is pointed out that China has not yet recovered from the effects of the opium habit that was foisted upon it by Western civilization, and that it is particularly unchivalrous of the United States Government to help foist a new evil upon this people while they are in the throes of reorganizing their society on a more intelligent and democratic basis.

DIFFERENTIAL DIAGNOSIS OF ABDOMINAL TUBERCULOSIS.

By GEORGE E. EBRIGHT, M. D., San Francisco.
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There are certain interrelations between the various forms of abdominal tuberculosis which lend themselves to brief discussion. From that standpoint it is possible to consider the several types of tubercular peritonitis, tuberculosis of the liver and gall bladder,—pancreas, spleen, fallopian tubes and ovaries and certain forms of intestinal tuberculosis including tuberculosis of the appendix.

While primary tuberculosis within the abdomen may be conceived as possible, it is safe to assume that there are few if any exceptions to the general rule that initial tubercular lesions are of the lymphatic tissues, particularly the lymph glands, and that when tuberculosis manifests itself as peritonitis or disease of the abdominal viscera, the same rule holds true that applies in pulmonary tuberculosis, that is, that the disease is a conflagration arising from a smoldering tubercular lymphadenitis which originated during infancy or in childhood. Von Pirquet maintained from his observations of the cutaneous tuberculin reaction that 60 per cent. of adults have or have had tuberculosis. Observations by subsequent investigators have more than confirmed this estimate. Fishberg (M. Fishberg, the extent of tuberculous infection in childhood among the children of New York tenements as evidenced by the application of the cutaneous tuberculin reaction, *Medical Record,* N. Y., 1915, LXXXVII, 417) found in 589 children examined at the age of 14 years 75% reactors; of 692 children of tuberculous parents in New York tenements at the age of 14, 83.79%, reactors; under 1 year of age 10% of children reacted.

Children are fortunately born with a high degree of resistance to all disease. For example, at birth a child's immunity to measles and to diphtheria is very high. Measles is rare under six months of age as is likewise diphtheria. This native resistance to disease diminishes rapidly after the first year, it being at a minimum between the ages of 5 and 7. It may be assumed that native immunity to tuberculosis is likewise high at birth and that infection by the tubercle bacillus is arrested by the first line entrenchments consisting of the lymphatic tissues. The tubercle bacilli, however, halted in the lymph glands produce adenitis which in the vast majority of cases is sooner or later checked by the protective formation of specific antibodies and the process becomes quiescent unless from one cause or another the individual's resistance is reduced and the activities of the invaders again become manifest resulting in various types of clinical tuberculosis. So that abdominal tuberculosis excepting lymphatic tuberculosis is to be considered in the strict sense always secondary.

The pathways by which tuberculosis gains a foothold in the peritoneal cavity are either the blood stream, lymph channels or the rupture of a lymphatic gland, from diseased fallopian tubes, appendix, gall bladder or other such focus. It has been demonstrated that the tubercle bacillus may pass through the intestinal wall without the presence of ulceration and also that the disease may spread apparently through the diaphragm through the thoracic cavity without apparent lesion, although it is questionable whether or not the circulating blood is not in this case the transporting factor.

Types of Peritoneal Tuberculosis.—Miliary tuberculosis of the peritoneum may be a part of miliary tuberculosis. Tubercular peritonitis proper presents itself in various forms depending largely upon the rapidity of the inflammation. In the more acute types are seen a considerable amount of fluid with little tendency to the formation of adhesions. In the more chronic cases of this ascitic form thickening and rolling up of the omentum takes place, thickening and shortening of the mesentery, thickening and shortening of the intestine.

In this type of tubercular peritonitis spontaneous recovery may take place. Elestrotov found that in 136 cases treated medically 31.6 recovered; in 240 cases treated surgically 78.3% recovered. (Osler's Modern Medicine, Vol. V, p. 578.) Again, a loculated or encysted form may be recognized. the intestines are matted together by adhesions and enclose collections of fluid which may be serofibrinous, turbid or purulent exudations, are larger becoming confluent masses of caseous material surrounded by adhesions and giving rise to suppurating foci among the coils of the intestines. In this way there may be multiple abscesses with a tendency to erode throughout the surrounding tissues either through the abdominal wall usually in the region of the umbilicus or through a viscus as the intestines, kidney, vagina. Finally, in the obliterative form universal adhesions join the viscera and the abdominal wall. There is no exudation as seen in the loculated or ascitic form. Sometimes the masses so formed are tumor-like and may be readily felt through the abdominal wall or through the rectum. As a matter of fact, in many cases of tubercular peritonitis a rectal examination discloses masses that with difficulty are felt through the abdominal wall. It is seen then that in the more rapid form there is little tendency to the formation of tumors and a considerable tendency to the formation of free fluid in the abdominal cavity and in the slow type the tendency to the formation of tumors is marked and practically no tendency for the extravasation of fluid and between these two types lies the intermediate form in which there is very considerable thickening and adhesion formation and pockets of extravasated fluid modified by caseous degeneration. The symptoms vary chiefly with the rapidity of the disease. There is usually a history of failing health, loss of appetite, strength and weight as is seen in any form of tuberculosis. In addition to this symptoms will vary according to the anatomical changes present. In other words, according to the position and density of adhesions and hyperplastic masses. Such symptoms will be referable to the alimentary tract and to pain arising from pressure or more often from pressure and stenosis of the intestines with resulting colic. It is not

* Read before the Medical Society State of California, Fresno, April, 1916.

unusual to find a leukocytosis, especially in children.

Tuberculosis of the Gall Bladder.—Two types have been recognized, a chronic ulcerative form associated with the presence of gall stones and an acute form with necrosis of the mucous membrane which may be associated with tuberculosis of the cystic and common bile ducts (Rolleston, Diseases of the Liver, etc., 1912, page 627). Primary tuberculosis of the gall bladder may occur as a chronic ulcerative process in the sense that it is not associated with clinical tuberculosis elsewhere. It may also occur following pyogenic infection of the gall bladder either with or without calculus. Secondary tuberculosis of the gall bladder may result from infection of other organs, especially the lungs or as part of general miliary tuberculosis (Bandelier-Roepke Die klinik der Tuberkulose, 1914, p. 440). The process may extend from the gall bladder to the liver or from the liver to the gall bladder. Diagnosis can scarcely be made except by means of exploratory laparotomy. Tuberculosis of the gall bladder may result in the persistence of a fistula after operation. Pericholangitis may be tubercular and shows miliary tubercles about the duct with an effusion of fluid and adhesions.

Tuberculosis of the female pelvic organs usually arises in the fallopian tubes, ovaries and uterus. It is of importance as a possible source of tubercular peritonitis. It is unnecessary to enter into a description of these lesions further than to say that small and large tubercles, ulceration and caseation with distension of the tubes and ovaries may occur. In relation to the treatment of tubercular peritonitis the removal of a tuberculous uterus or the adnexa, particularly a tubercular fallopian tube, may be the controlling factor in the treatment of peritoneal tuberculosis. This is also true regarding tuberculosis of the gall ladder and appendix and these three organs, appendix, fallopian tube and gall bladder should always be carefully inspected during an operation for tubercular peritonitis.

Tuberculosis of the liver may be either a part of general miliary tuberculosis in which case the lesions are diffuse throughout the liver or localized tuberculosis of the liver may occur in the form of bile duct tuberculosis forming caseous tuberculous masses or solitary tuberculous abscess, the latter of which is very rare. As a rule hepatic tuberculosis is secondary to outspoken processes elsewhere, particularly tubercular ulceration of the intestines in which condition hepatic involvement is rather more common than is generally suspected; the infection taking place through the portal vein produces miliary tubercles and later masses of tubercular tissue,—in effect a tuberculosis of the portal spaces. The liver is usually somewhat larger than normal and on section shows a number of white caseous areas or bile stained cavities with caseous walls. Jaundice is absent. Again, multiple tubercular lesions may occur in the liver closely resembling gummata and fairly easily nucleated. Sometimes these masses may be felt through the abdominal walls during life and may be associated with considerable pain. In some cases splenic enlargement is present. Resemblance to carcinoma of the liver is very considerable on account of emaciation, general weakness and loss of appetite.

With secondary infection of pyogenic organisms the tuberculous mass may develop into an abscess which may be either within the substance of the liver or near the surface, the latter case giving rise to localized perihepatic or subphrenic abscess. Single lesions are very rare. There is nothing characteristic in the symptomatology of tuberculosis of the liver or gall bladder and diagnosis is practically never made except upon operation or post mortem.

Tuberculosis of the Pancreas.—Miliary tubercles in the pancreas are not uncommon in tuberculosis of other organs, especially in children. But while tubercular masses have been described in the pancreas palpable through the abdominal wall, they are most rare and appreciable interference with the function of the pancreas by tubercular disease has not been described.

There are three interesting forms of tuberculosis which may occur in the region of the caecum,—tuberculosis of the appendix, hypertrophic tuberculoma of the ilium and hypertrophic tuberculoma of the caecum and tubercular lymphadenitis of the mesentery behind the appendix. The latter may give rise to such large glands as to stimulate chronic appendicitis. As a rule they are readily removed. Tuberculosis of the appendix is said to be found in 2% of the operations upon the appendix (Lockwood). It is usually secondary to tuberculosis of the caecum but may be primary. Usually there is simply tubercular ulceration or in other cases extensive involvement of the appendicial walls with caseation. Milary tubercles and adhesions may be seen in the adjoining peritoneum either localized or more diffused. Hypertrophic appendicial tuberculoma has been observed. Macroscopically it is many times difficult to distinguish tubercular from other forms of appendicitis. Hypertrophic tuberculoma of the ilium or caecum arises from either submucous or subserous lesions resulting in great thickening of the intestinal wall to such an extent that hyperplastic tuberculoma of the ilium or caecum may readily be mistaken for sarcoma. A helpful point in the diagnosis of tubercular appendicitis and hypertrophic tubercular changes in the ilium or the caecum lies in the fact that tuberculosis of the lung is also present. The local symptoms are no different from those of other forms of tumor or low grade inflammation. Stierlin's sign is of importance in differentiating hyperplastic tuberculoma of the caecum from cancer. By means of the X-ray it is seen in the presence of the tubercular condition that the bismuth mass goes more rapidly through the caecum resulting in an X-ray picture which gives no bismuth shadow in the caecum—only above and below it. If the question of diagnosis between hypertrophic tuberculoma of the ilium and of the caecum arises, the caecum empty of bismuth with the X-ray test would speak in favor of involvement of the caecum or on the other hand if bismuth shows in the caecum it would speak in favor of the lesion being in the

ilium. Strangely enough Stierlin's sign is absent in cancer of the caecum. Patient R. D., 29 years old, tailor. Father died of tuberculosis. Patient came to the University of California service, San Francisco Hospital, suffering from pain in the right iliac region. He had been treated for pulmonary tuberculosis which was of about one year's duration. His present illness began with a sudden pain in the right lower quadrant which disappeared in a few minutes some two and one-half weeks before admission to the hospital. This pain recurred suddenly while walking and was accompanied by nausea and vomiting. The pain spread to the whole abdomen. The patient was admitted to the hospital with a fever of 100.5°. Examination of the abdomen showed slight rigidity of the entire abdomen, lower more than the upper, and of the right more than the left, considerable tenderness in the outer half of the right iliac fossa. An elongated round boggy mass could be palpated at McBurney's point, which was very tender. With rest in bed the pain and local symptoms disappeared in the course of a week so that there remains at the present time a patient with pulmonary tuberculosis and a mass about 2½ inches long by an inch wide, sausage-shaped, lying obliquely in the region of the appendix only slightly tender on pressure and which undoubtedly is a hypertrophic tuberculoma either of the ilium or of the caecum. Stierlin's sign being present in this case we take it to be a lesion of the ilium.

Chronic hyperplastic tuberculosis while probably more common in the iliocæcal region has also been observed in the sigmoid flexure. The diagnosis of subserous fibroma should be avoided until tuberculosis has been excluded.

In conclusion it may be said that tuberculosis of the abdominal viscera is commonly associated with pulmonary tuberculosis but that the primary infection is to be searched for in the lymphatic system, especially the peribronchial and retroperitoneal glands; that the type in which tubercular peritonitis presents itself depends upon the rapidity of the inflammation, rapid processes being associated with tendency to formation of fluid and less tendency to the formation of adhesions a slower process giving rise to thickening and adhesions with fluid in the shape of walled-off collections and abscess formation, the most chronic giving rise to adhesions alone and hyperplastic growths in the walls of the intestine. Fever is usual in the acute and subacute forms and may be absent in the chronic forms, or there may be a subnormal temperature. That the fewer adhesions the better prognosis and that the ascitic form lends itself readily to operative interference. When operation is resorted to special attention should be devoted to the appendix, fallopian tubes and gall bladder.

TOXIC GASTRIC HEMORRHAGE.*

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While the development of gastric surgery has proved ulcer to be the most frequent cause of bleeding from the stomach and duodenum, the coincident development of pathology in the living has emphasized the fact that frequently there may be gastric hemorrhage without any demonstrable surgical gastric lesion. The opinion is prevalent among the laity that hematemesis means ulcer requiring operation, and hemorrhage from the stomach is too readily accepted by physicians as sufficient evidence to warrant surgery. I wish to call attention to the hemorrhages occurring from other than true surgical lesions, and to the importance of differentiating the causes of bleeding that are medical from those that are surgical.

The calloused ulcer derives greatest benefit from surgery. However, gastric surgery has been too often resorted to without benefit to the patient. Particularly is this true in cases in which hemorrhage was the principal cause for exploration. Often when an abnormal constitutional condition is not obvious, bleeding from the upper gastro-intestinal tract is considered as coming from a so-called hidden or non-symptomatic chronic ulcer, and the patient carries a gastro-enterostomy for ulcer for which there was not sufficient evidence before operation and no evidence at the time of operation. The burden of differentiating hemorrhage due to chronic ulcer from hemorrhage due to non-surgical conditions rests with the internist.

It is true that we occasionally see ulcers, benign and malignant, of which the histories are meager and alone are not sufficient for clinical conclusion The proportion of these will decrease with a more general knowledge of the varying clinical factors that are helpful in the recognition of the atypical group, and roentgenology will further assist in their diagnosis. Clinical study supplemented by the diagnostic efficiency developed in gastric roentgenology has made it possible to determine the presence or absence of the bleeding gastric lesions that can be benefited by surgery in a very large percentage of the cases of hemorrhage from the stomach.

To designate the oozing of blood from the stomach in the supposed absence of chronic ulcer, Sir Edwin Cooper Perry suggested to Hale White [1] the term "gastrostaxis," which is similar etymologically to "epistaxis." White [2] advanced the opinion that there might be a clinical group of this type among young women having pain, vomiting, and hematemesis, without ulcer symptoms, and in whom spontaneous recovery was the rule. The suggestion brought out considerable discussion in regard to gastric hemorrhages of obscure origin by White,[3] Bolton,[4] and Hort.[5] As the conditions in which such hemorrhages usually occur are toxic, the term "toxic gastric hemorrhage" suits our purpose better and will be used in referring to them here.

Blood that is vomited and tarry stools do not always mean hemorrhage from chronic gastric ulcer. Blood from the lungs and pharynx may be swallowed and later vomited. Bleeding from

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esophageal varices, particularly when associated with the toxic state in cirrhosis of the liver, may be severe and have the appearance of gastric hemorrhage. In the purpuras, leukemias and anemias, especially anemias associated with enlargement of the spleen and liver, there may be severe bleeding from the stomach. In constitutional diseases and toxemias associated with hepatic and renal disease, it is common to find on necropsy that the gastric mucous membrane is intact, though vomiting of blood occurred during life. Blood may be vomited during exacerbations in states of hypertension and in secondary congestions of the liver and spleen. Endocarditis may be a remote cause of gastric hemorrhage. Also, exudative erythemic states of the viscera are possible causes of bleeding.[6] Arteriosclerosis of the abdominal vessels with aneurysmal dilatation and rupture into the stomach has been reported.[7] During the attacks of gastric crises in tabes, there is often coffee-ground vomitus and at times bleeding may be severe. Young females may have extensive hemorrhages with no proof of chronic ulcer and with usually spontaneous recovery.

With infections of the gallbladder and appendix there are occasional hemorrhages for which no adequate cause in the stomach is found at operation. In an operated series studied in the Mayo Clinic,[8] bleeding was associated with infections of the gallbladder and gastric symptoms in 5 per cent., and with appendicitis and gastric symptoms in 2 per cent. Deaver[9] mentions infections in the fallopian tubes as a causative factor in some cases of gastric hemorrhage. Bleeding from follicles or superficial erosions in the stomach permitting hemorrhage may be secondary to acute infections in the tonsils. Rosenow[10] has shown the association of various streptoccoccic infections with bleeding from hemorrhagic points and superficial ulcers in the gastric mucosa. These may become so extensive that large patchy areas of the mucosa ooze blood, though when wiped off, individual points are made out with difficulty.

Dieulafoy[11] has called attention to gastric bleeding from the two following varieties of non-chronic ulcer: (1) Simple erosions consisting of mere abrasions of the surface epithelium. These though so small as to be scarcely perceptible to the naked eye, may give rise to most alarming hemorrhages. At necropsy they may be easily overlooked, but during the course of the hemorrhage the mucous membrane appears to be studded with numerous bleeding points. (2) Exulceratio simplex. The lesions of the type to which Dieulafoy applies this term rather more extensive, and the surface layers are removed to such an extent that the arterioles running under the muscularis mucosae are exposed. This form may give rise to severe hemorrhages that may even prove fatal. On operation the condition appears as small bleeding fissures, small patchy areas oozing blood, or thick hemorrhagic infiltrations from which blood literally seeps.

Deaver,[9] in discussing hemorrhagic disease of the stomach not associated or closely related to gross ulcer, says: "Excepting extrinsic poisons, I believe the violent congestion of the gastric vessels is primarily dependent on an intra-abdominal, or more rarely, remote, focus of infection." This focus of infection he believes is most commonly the appendix or gallbladder. According to Mayo Robson[12] the gastric lesions after death in some cases of sudden severe hemorrhages, particularly in the young in whom there is no clinical evidence of ulcer, seem altogether inadequate to explain the nature of the serious hemorrhage. It is his opinion also that: "Capillary oozing or bleeding from arterioles is much more common and accounts for many more cases of gastric hemorrhage than has been hitherto supposed." Hemorrhages of this kind, parenchymatous hemorrhages in the apparently healthy male, hemorrhagic gastralgias and the large group of variously defined bleeding from the stomach in which there is sudden onset, absence of symptoms and usually spontaneous recovery, are of infective or toxic origin, and surgery will be of doubtful benefit.

Typical acute gastric ulcer may be the source of repeated hemorrhages if there is erosion of the vessels at its base, but rarely causes fatal bleeding. At operation these ulcers may be shallow and not visible or palpable through the wall of the stomach. When secondary to gross infection elsewhere, mucous ulcers may be multiple. Chronic gastric and duodenal ulcers as a rule do not bleed copiously. Bleeding from malignant disease usually is small in amount like that from ulcer, and more or less continuous.

In seeking a cause for gastric hemorrhage a history of ulcer should be sought and, when necessary, diagnostic evidence should be brought out by every adjunctive means available. If evidence indicating ulcer is not strong, effort should be made to prove or exclude all the numerous conditions that might be underlying causes of the hemorrhage.

In general, surgery offers the best results for ulcer of the chronic calloused type. In this condition the symptoms of ulcer are marked and the patient goes to the physician usually because of the distress from the ulcer rather than because of the hemorrhage. A second group of patients with gastric hemorrhage lays greater emphasis on the bleeding and complains only of gastric symptoms that are more or less indefinite. In such cases effort must be made to determine the presence or absence of calloused ulcer or of a toxic or infective condition as the cause of the hemorrhage. A third group of patients are those who come for examination because of the hemorrhage but whose gastric symptoms are of minor importance. They believe that they have an ulcer, frequently have been told that they have ulcer, and have often resorted to gastric surgery which has not prevented subsequent hemorrhages.

Speaking broadly, cases of hemorrhages without gastric symptoms, either before or after surgery, should be considered toxic. The non-surgical nature of the causative factors, such as the blood diseases and secondary congestions, are usually easy to determine. Medical observation will further develop the exact nature of many hemorrhages of the toxic group and relegate them to their proper medical sphere. When an

infective source, either intra-abdominal or remote, is found, the possibility of an association between the gastric hemorrhage and this infected atrium should be considered. In many such cases the hemorrhage is probably toxic. When medical observation determines the presence of abnormal constitutional states such as diseases of the blood, renal toxemias, disproportional varices, pathologic vascular tensions, multiple angiomas, syphilis or tuberculosis, the hemorrhage should be considered of toxic nature. Correction, when possible, of the underlying conditions, and waiting, rather than surgery, is advisable. Any surgery in these cases should be only in the nature of exploration without promise as to results. Spontaneous hemorrhages occuring in young women and parenchymatous hemorrhages in which evidence does not point to chronic ulcer, should also be considered toxic. They are not surgical; spontaneous recovery is the rule.

Patients giving a history of repeated severe hemorrhages over many years, with an ulcer history, which though not clear-cut, is strengthened by adjunctive evidence, and in whom no constitutional cause can be found, should have an exploratory operation. In such cases the hemorrhage is probably due to a surgical condition. Well-nourished patients who have had one or a few hemorrhages, and for whom the clinical or contributory data are very poor, may be considered toxic until medical observation proves the absence of a constitutional condition as a factor, or time brings out evidence of ulcer. If surgery is indicated at all for gastric hemorrhages occuring in the presence of intra or extra-abdominal infection, and accompanied by indefinite gastric symptoms, it should be applied to the focus of infection rather than to the stomach.

Patients who have had repeated hemorrhages for many years, who are past middle life, whose general appearance is below par and of whom the history and evidence of ulcer is indefinite, should also be carefully studied for toxic causes. The following abstracts of histories will serve to illustrate various types of gastric hemorrhages.

CLINICAL EVIDENCE ALONE INSUFFICIENT FOR ULCER. EXPLORATION BECAUSE OF POSITIVE ROENTGEN FINDINGS.

Case 146,581, M. C. B., traveling salesman, aged 52 years. Examined November 30, 1915. Patient had been having hemorrhages from the bowel, black and tar-like, for thirty years; in all, about twenty; in bed after each attack. The last hemorrhage occured in June, 1915. Three hemorrhages in 1913. No clear-cut gastric history, though he had had distress for days at a time, and on a few occasions for a couple of weeks. Food relief variable; never used soda; never had colic. Present trouble with stomach of about two weeks' duration. He dieted for two years but did not obtain relief. In present attack he had had a little distress at night, varying from 11 p. m. to 5 a. m., and some distress after meals. Most of the hemorrhages "have come out of a clear sky when he was feeling his best." Epistaxis frequent in youth, but never put him to bed.

Examination. A healthy looking man, 5 feet, 9 inches in height, weighing 145 pounds; no weight loss. Mucous membranes somewhat pale. Multiple pea-sized, raspberry angiomas over body. Hemoglobin 70 per cent. Gastric analysis: Acids 58, 44, 14; no food remnants. Wassermann negative. Coagulation time three minutes. Blood pressure 128, 90. Eye grounds negative. Urine negative. Protoscopic findings negative. Roentgen findings: Cap deformity; duodenal ulcer.

A history covering thirty years with slight symptoms except for bleeding, clinical absence of obstruction at the pylorus, and the angiomas over the body, made diagnosis doubtful. An exploratory operation was performed because of the roentgen evidence. A duodenal ulcer was found one-half inch below the pylorus.

CLINICAL AND ADJUNCTIVE EVIDENCE INSUFFICIENT FOR ULCER. CONSIDERED TOXIC AND NOT NOW SURGICAL.

Case 141,079; T. A. C., advertising manager of a newspaper, aged 50 years. Examined September 15, 1915. Patient had been in the habit of eating fast; he worked hard; burned the candle at both ends. Formerly a printer. Twenty-five years ago he had anemia which he thought was due possibly to lead poisoning. In the last twenty-five years he had had a few attacks of gastric trouble lasting a week or two. Symptoms meager. In October, 1914, he fainted one day while in the toilet; soon vomited food and blood. Vomited clots three times within a short period. Had tarry stools for the following three or four days. Was put on a Von Leube diet and rapidly regained his health; well since. Came for examination eleven months later because an ulcer had been diagnosed at the time of the hemorrhage. No symptoms since hemorrhage except a little discomfort without food relation, in the left hypochrondrium.

Examination. Height 5 feet, 10½ inches; weight 195 pounds; no weight loss; skin somewhat highly colored. Blood pressure 178,100. Gastric analysis: Acids 66, 56, 10. No food remnants. Hemoglobin 89 per cent. Differential blood count normal. Coagulation time eight minutes. Wasserman negative. Negative fundi. Urine showed a few hyalin casts. Roentgen finding: Stomach indeterminate.

On account of lack of evidence of ulcer, and because of the blood pressure, urine findings, coagulation time, and patient's generally well-nourished condition, diagnosis was made of toxic hemorrhage. He was advised about caring for his general health, and sent home for observation.

NEGATIVE GASTRIC EXPLORATION. CONSTITUTIONAL STATE. VISCERAL ANGIO-NEUROTIC EDEMA BELIEVED TO BE CAUSE OF BLEEDING.

Case 101,588; W. H. S., paper hanger, aged 40 years. Examined March 4, 1914. This patient had been urged a number of times to have operation for gastric ulcer. He had gastric trouble intermittently for twenty-three years. When 17 had cramps, were so severe as to double him up. He used to tie a towel tight around his waist, and a number of times was rolled over a barrel to relieve the cramping pain. These attacks came frequently for a few weeks and then disappeared for weeks or months. Only occasional trouble between the ages of 20 and 30. In an attack when about 31, he vomited a large handful of clots of blood. No clear-cut food relation to gastric pain. Present attack three weeks. No regularity. For three months had had most marked angioneurotic disturbances. Large plaques came out on his skin each night. Roentgen findings: "Lesion of the stomach at or near the pylorus." The patient had angioneurotic swellings. At exploration (C. H. Mayo) neither lesion nor cause for hemorrhage

was found in the stomach. The gallbladder, showing doubtful pathology, and the appendix were removed.

The attacks of pain have continued since operation without any material change in nature. They are regarded as visceral manifestations of angioneurotic edema. Because of the constitutional condition the hemorrhages may be considered toxic.

YOUNG WOMAN. HEMORRHAGE. TREATMENT FOR ULCER. NO SYMPTOMS OR EVIDENCE OF ULCER. SPONTANEOUS RECOVERY.

Case 147,636; Miss A. R., aged 21. Examined December 11, 1915. She complained of gastric trouble and menorrhagia. Eighteen months before a severe hemorrhage occured in the stomach; melena persisted for a number of days. She was put to bed and kept on milk diet for seven months. Her physician said the bleeding came from an ulcer in the stomach. She had been away at school, had eaten irregularly injudiciously a large amount of candy; enjoyed social activity, dancing, tennis, etc. Indefinite distress in the stomach began a month or two before the hemorrhage, about the middle of the school year. Stomach always tender; with pain and soreness after eating ordinary foods; never free periods. No food relief. Menstruation irregular, increased flow for three years; for two weeks at each period a profuse flow. Patient said she had had grippe three or four times. Wrenched her back four years ago and it "had been sore ever since."

Examination. A thin, somewhat pale young girl. Very nervous. Weight 104 pounds. Blood pressure 100, 78. Urine negative. Hemoglobin 89 per cent. Gastric analysis: Acids 20, all combined. No food remnants. Roentgen findings: Stomach negative. At the hotel the patient ate everything, and forgot all about her stomach.

Because of the hemorrhage she firmly believed that she had ulcer, as did her mother and her brother, a physician. Her physician had advised operation for ulcer. The seven months in bed on ulcer treatment had made her a marked neurasthenic. There was neither clinical nor laboratory evidence of ulcer. Patient was told that there was no evidence of chronic ulcer and operation was not advised.

GASTRIC ATTACKS WITH HEMORRHAGES, FOLLOWING TONSILLITIS.

Case 145,217; Mrs. R. T., aged 24 years. Examined November 8, 1915. Epigastric pain, hemorrhages from stomach. She has had repeated attacks of tonsillitis and rheumatism. Distress in stomach at times for eight years. Cramp-like pains lasting five or ten minutes associated with nausea. Vomited during the first five years, but seldom vomited food. Has had numerous hemorrhages from stomach, in one of which she said she lost a quart of blood. Gastric symptoms lasted ten days to two weeks; she was then relieved for a period of months. No definite food relationship. The pain was present even when a strict diet was maintained. About three attacks of tonsillitis each winter. Gastric attacks always followed tonsillitis. In a remission four weeks before there was a hemorrhage from the stomach.

Examination. Short, soft, systolic blow heard at cardiac apex. Blood pressure 122, 82. Pulse 68. Temperature 99.2. Urine negative. Hemoglobin 70 per cent. Gastric analysis: Acids 48, 28, 20. Roentgen findings: Chest negative; stomach indeterminate.

Consultant's Note. "Not typical ulcer. History suggests superficial type of acute lesion with hemorrhage. The frequency with which tonsillitis has preceeded attacks is interesting." Tonsillectomy by Dr. Matthews. Culture from tonsils by Dr. Rosenow showed streptococci, and animals injected showed multiple hemorrhages and superficial ulcers of the stomach, due to streptococci. Hence these hemorrhages, which seemed to be toxic, were apparently due to localized hematogenous infections of the mucous membrane of the stomach, following tonsillitis.

GASTRIC SYMPTOMS WITH HEMORRHAGES. EXPLORATION: CHRONIC APPENDICITIS. NEGATIVE GALLBLADDER AND STOMACH.

Case 126,387; E. G. P., contractor and builder, aged 46 years. Examined March 12, 1915. This patient had had myalgias, followed by ecchymoses that put him in bed for three or four days. For fifteen years he had had trouble with his stomach which came on in spells formerly lasting for months. Trouble now continuous. Had had some food relief and relief by alkalies and vomiting. A burning sensation was felt in the stomach usually from 10 to 11 a. m. Hemorrhages from the stomach. Vomited a large quantity of blood during a period of two days. Four months ago had another hemorrhage and at that time tarry stools. Described pain radiation as being "most anywhere" in the upper abdomen. Patient very nervous; continuous headache for last two months.

Examination. Under weight. Blood pressure 150, 105. Hemoglobin 88 per cent. Coagulation time five minutes. Wassermann negative. Moderate right pyelitis, proved by ureteral catheterization. Gastric analysis: Acids 34, 22, 12. No food remnants. Exploration for peptic ulcer (W. J. Mayo).

Findings at operation: "Sub-acute appendicitis. Appendicitis apparently would account for symptoms as it was rather unusually well marked. Gallbladder and duodenum normal. Gallbladder somewhat adherent, but empties easily and contains no stones." Appendix removed. After negative exploration of upper abdomen, the hemorrhages were believed to be gastrotoxic, secondary to the infection in the diseased appendix.

GASTRIC SYMPTOMS, HEMORRHAGE, CAME FOR "ULCER." EXPLORATION: CHOLECYSTITIS PANCREATITIS. NEGATIVE STOMACH AND DUODENUM.

Case 124,020; Mrs. N. M., aged 36 years. Examined February 8, 1915. Trouble with stomach began four years ago. First attack, four months. One free period of three years. Second attack began one year before; was ill nine months. She had pain nearly continuously except when eating. Vomited sour water. Once she had delayed vomiting. Slight food relief. Soda relief formerly. Had sharp cutting pain in left epigastrium for which her physician had given morphia. Tarry stools. Coarse and sour foods caused distress. Patient referred to the Mayo Clinic with a diagnosis of ulcer with hemorrhage.

Examination. A very neurotic, fairly well nourished woman. Area complained of the left epigastrium. Slight tenderness to deep pressure in right lower abdomen. Blood pressure, 125, 80. Urine negative. Hemoglobin 85 per cent. White blood cells, 7000. Gastric analysis: Acids 28, 12, 16. No food remnants. Roentgen findings. Stomach indeterminate. The physician at home and the patient were sure that ulcer was the cause of her distress and bleeding. She was under observation a week, then sent for exploration of the stomach, gallbladder and the appendix. Cholecystectomy and appendectomy were done.

Findings at operation: (E. S. Judd) "Definite chronic cholecystitis and chronic pancreatitis. Head of pancreas twice its normal size, chronic appendicitis; stomach and duodenum negative."

NEGATIVE EXPLORATION—TOXIC CONSTITUTIONAL CONDITION—CARDIO RENAL SYNDROME WITH TRANSIENT HYPERTENSION.

Case 135,561; H. R. T., bank cashier, aged 60 years. Examined July 17, 1915. Patient had had hemorrhages from the bowels (tarry stools) seventeen years, ten years and five years ago. In September, 1914, also vomited blood; collapsed. In years past had to be careful of his diet and had intermittent discomfort. A clear history was difficult to obtain. A diagnosis of ulcer was made after each hemorrhage. In the Fall of 1914 a gastrojejunostomy was done for "ulcer on anterior duodenal wall one inch below the pylorus." Four weeks before coming for examination he had collapsed and the next day had profuse tarry stools.

Examination. Five feet, seven inches in height; weighed 130 pounds; underweight. Appeared weak and had marked pallor. Some bagginess under eyes. Sclera pearly; looked nephritic. Heart five inches to left. Diastolic murmur at aortic area. Blood pressure 185, 85. Hemoglobin 45 per cent. Coagulation time six minutes. Differential blood count normal. Gastric analysis: Acids 64, 54, 10. No food remnants. Wassermann negative. Old patches of hemorrhages in fundi. Phenolsulphonephthalein functional test 43 per cent. in two hours. Roentgen findings. Gastroenterostomy functioning; otherwise negative. Patient gained on ulcer diet. Was kept under observation for three weeks. Believed to be gastrotoxic, but because of reported presence of ulcer at the time of gastroenterostomy done elsewhere, and in order to clear up the nature of the condition, an exploratory operation was performed (W. J. Mayo).

Surgical report: "Two inches of the stomach and two inches of the duodenum were resected. Gastro-enterostomy in good condition. Site of supposed ulcer on duodenum resected. Patient in wretched condition." Pathologic report: Pyloric ring of stomach normal; on section scar of ulcer could not be found.

The patient gained rather slowly after operation. About three months later he had a very severe hemorrhage. Was found unconscious in a pool of blood in the bathroom. A letter from his home physician stated that a blood pressure of 250 systolic had been recorded a few days before the hemorrhage. It is probable that all of the bleedings were the result of a constitutional state, toxic in type, and of a nature that surgery could not benefit.

SUMMARY.

Toxic gastric hemorrhage is essentially a medical condition. Hemorrhage does not always mean chronic ulcer. Surgery should be resorted to for the calloused type of ulcer, and for this type only will it give the best results. Recognition of the true cause of hemorrhage from the upper gastro-intestinal tract is sometimes most difficult. At times evidence will warrant exploration to prove or exclude peptic ulcer as a cause. The presence of a constitutional disease without sufficient evidence of ulcer, makes medical observation and study, rather than surgery, advisable. In cases of hemorrhages of obscure origin, search for infected foci should be made and the possibility of their association with the cause of the hemorrhage should be considered. In addition, studies of blood diseases associated with bleeding, and further studies in blood pressure, with recognition of transient hypertensive states, will help to define and separate hemorrhages having their origin in surgical ulcer from gastric hemorrhages of acute infective and toxic origins.

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HYPOPHYSEAL SYMPTOMATOLOGY; A REVIEW.*

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Recent conceptions of the ductless glands differ from previous ideas, chiefly in that observers are now beginning to recognize the inter-dependence of these organs; to consider them more as inseparable complexes; and are striving to estimate correctly both their individual and reciprocal influences. The clinical manifestations resulting from endo-secretory disorders are usually described as composing one or more phases of a general polyglandular syndrome. It is the purpose of this paper to consider, very briefly, certain influences which the pituitary body may play in the production of some of these symptom-complexes.

In 1838, Rathke [22] described an ectodermic pouch budding from the bucco-pharangeal cavity which meets and partly surrounds the infundibular prolongation of the anterior cerebral vesicle. The tip of this prolongation subsequently becoming thickened, is known as the infundibular body, neurohypophysis or pars nervosa. Later the developing sphenoid bone obliterates the lumen of Rathke's pouch, the tip of which envelopes the infundibular body. This combined neuro-epithelial structure, or pituitary body proper, later becomes endowed with a dural capsule and occupies the sella turcica. It is present in all vertebrates showing its phylogenetic importance.

Histologically, the gland is divided into three parts—(I) the anterior lobe (pars anterior, pituitary gland proper) consisting of columns of cells surrounded by large sinusoidal spaces. These cells are classified by Flesch [10] (1884) into so-called chromo-philes, composed of eosino-philes and basophiles; and chromo-phobes or neutro-philes. These cells show hypertrophic changes during pregnancy; atrophic during hibernation, etc.; (II) the posterior lobe (pars nervosa, infundibular body, neuro hypophyses, etc.) composed of a meshwork of loosely placed neurologia whose fibres radiate toward the infundibulum; (III) the pars intermedia of Herring, or the epithelial investment of the infundibulum. The two become fused. Structur-

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ally, the anterior lobe suggests a typical endocrinous gland discharging (according to Thaon [25]), a stainable colloid into the blood stream. Hyperplasia of this lobe apparently stimulates tissue growth, skeletal, cuticular, and subcuticular, and exerts an excitatory influence on the sex glands. The posterior lobe probably secretes an exceedingly soluble colloid which seems to have a definite effect on tissue metabolism, etc., directly into the cerebrospinal fluid by way of the third ventricle. This has been fixed and demonstrated by Herring [17], 1908. The blood supply, from an embrological standpoint, is parodoxical, the anterior lobe being supplied by a number of small arterioles passing down the infundibular stock, while a single artery coming from below and behind supplies the posterior lobe.

Hypophyseal physiology is slowly being unraveled. It was the Galenic and Vesalian [26] view that the gland elaborated a mucous secretion (pituita) which entered the nose and lubricated the nasal cavities. Magendie [27] considered it a lymph gland which collected the cerebral lymph and distributed it to the circulation.

Recent methods of investigation, (I) by injection of extracts; (II) by feeding experiments; (III) by glandular transplantation; (IV.) by partial extirpation of the hypophysis, have thrown much light upon its physiology.

(I.) The following effects have been recorded by the injection of extracts (whole gland): Houssay [27] thinks all extract "substances are secreted by the cells of the pars intermedia, and then collected and concentrated or changed into more active forms" in the posterior lobe. Oliver and Schäfer [21] (1895) noted a rise in blood pressure due to vaso-constriction and augmentation of heart beat; Howell [18] (1898) noted that posterior-lobe extract slowed and strengthened the pulse; Dale [8] (1906) called attention to its effect upon uterine contractions; Schäfer and Herring [24] (1906) showed that posterior lobe extracts produce kidney dilatation and diuresis (acting on the renal epithelium directly). Bell and Hick [2] (1909), Dale [8] (1909), Frank-Hochwart and Frohlich [12] (1910), noted that it caused vesicle and intestinal contractions; Borchard [4] (1908), Goetsch, Cushing and Jacobson [7] (1911), noted that it lowered the assimilation limit for carbohydrates and caused glycogenesis. Repeated subcutaneous injections of sterile extracts of the whole gland or posterior lobe alone cause emaciation. (Fodera and Pitten,[11] 1909) (Crow, Cushing and Homans,[6] 1910.) Clinically, posterior lobe extract has been used to strengthen uterine contractions, to increase intestinal peristalsis, and to stimulate the heart, etc.

(II.) Feeding Experiments—Goetsch's [14] (1916) recent "Studies on the Influence of Pituitary Feeding upon Growth and Sexual Development" in rats is most enlightening. He concludes whole gland, when fed in excessive doses, causes failure to gain in weight, loss of appetite, increased peristalsis, mild enteritis, certain nervous manifestations such as weakness of the hind limbs and muscular tremors. These symptoms are probably due to the posterior lobe element alone, for they can all be produced by feeding posterior lobe but not by feeding anterior lobe extract. The influence on the female sex glands of whole gland feeding shows ovaries, tubes and cornua of uterus larger and more vascular than in control. The ovary matures sooner, fimbriated end of tube is farther developed, lining cells are more ciliated and active, and endometrium is hypertrophic; likewise the uterine muscle is considerably more thickened and vascular than in the control.

Influence upon male sex glands (whole gland); —Testes show earlier growth and development; are completely and permanently descended at an earlier date, weigh more and show etxremely spermatogenesis when the control animal is still sexually immature.

Feeding anterior lobe alone (Goetsch) shows increase in weight, greater and more vigorous bodily growth, earlier and more active sexual development (shortened 1/3 normal time), sexual instinct early awakened, breeding earlier and oftener, the female having two pregnancies in seven months as compared to none in the control animals; and sexual organs in both sexes show histological advancement in all sex elements in the fed animals. The effect lasts through the life of the animal and even exerts a stimulating influence on the offspring in intrauterine life and during lactation.

Feeding of posterior lobe extract alone inhibits sex development and causes loss in weight, mild enteritis and increased intestinal peristalsis. Ovarian extract (corpus luteum) has a stimulating effect upon the female, and a retarding influence upon the male sexual development. These results are cited with some emphasis because of new hope thus offered clinically by glandular feeding in hypopituitary states.

(III) Glandular transplantation (experimental hyperpituitarism) has been repeatedly tried and has universally failed. Halsted's [16] so-called "physiological deficit" must be present—i. e., hypopituitarism in these cases, if one is to expect reasonable success. Clairmont and Ehrlich [5] (1909) transplanted into the spleen; Schäfer [23] (1911) transplanted subcortically, subcutaneously, intromuscularly, into peritoneal cavity and into kidney substance with only a transient glycosuria. Waitz felder [28] 1914 reports an isolated attempt to produce hyperpituitarism in man by transplanting a pituitary and pineal gland (taken from an executed criminal) into the pectorals of a patient suffering from hypopituitarism. His attempt failed—marked dispositional changes ensuing for a short time only.

(IV) Extirpation methods:—(Experimental Hypopituitarism). Cushing [7] and his associates have succeeded in producing constitutional disturbances in animals by partial hypophysectomies, which simulate and explain some of the clinical syndromes observed in man, and which give just the opposite picture produced by Goetsch's feeding experiments. Cushing showed that hypophysectomized adult canines developed widespread adiposity, nutritional

changes of skin and its appendages, disturbance of carbohydrate tolerance, lowering body temperature, sexual inactivity, atrophy of the sexual glands and modification of most of the other ductless glands proved histologically. Hypophysectomized puppies remained infantile through life. Hypophysectomized adults showed regressive changes. These experiments proved that certain recognized symptom-complexes result from lessened pituitary glandular activity which is confined chiefly to the anterior lobe. Inversely, after castration in animals, the anterior lobe of the hypophysis becomes hypertrophic; and after pancreatectomy, definite changes appear in the posterior lobe, etc.

Clinically, pituitary disorders are usually classified under three heads:—the hyperpituitarism seen in gigantism or acromegaly; hypopituitarism evidenced by infantilism or sexual regression in adults; and dyspituitarism, or a mixture of the above types.

The hypophyseal diseases may be aptly compared to allied thyroid disturbances, in which the relation of hyperthyroidism to Graves' disease is similar to that of hyperpituitarism to gigantism or acromegaly; and in which hypothyroidism with its prototypes of cretinism or Gall's myxoedema is comparable to hypopituitarism with resulting infantilism or adult sexual regression; and lastly, as alternating periods of thyroid overactivity and sluggishness result in dysthyroidism (Marine's view)—so the same phases of alternating hyper- and hypopituitarism gives rise to a more complex state—dyspituitarism.

Hyperpituitarism gives rise to two similar but distinct pictures. If it sets in before epiphyseal ossification is completed—gigantism results. This syndrome is described as the "typus Lannois."[19] If the process occurs in adult life acromegaly follows. This is known as the "typus Marie" from the one who first described acromegaly in 1886.

Hypopituitarism is characterized (a) by a persistence of both skeletal and sexual infantilism when the process occurs in childhood—i. e., so-called "typus Frölich"[13]; and (b) by adiposity with reversive sexual changes when it originates in the adult, producing a clinical syndrome, the analogy of which is seen in Cushing's hypophysectomized dogs.

Dyspituitarism may combine all the varying clinical phases evidenced in primary hyper- and hypo-pituitary disease. Accordingly the largest number of cases fall into this class.

The following case of acromegaly was studied by the author under the direction of Dr. Harvey Cushing some two years ago.

The patient, male, was a healthy baby, oldest of six children, parents large boned. At thirteen, he began to grow tall and weighed 200 lbs.; was noted for feats of physical strength, was intelligent, had uncontrolled libido sexualis. At twenty-three, he had a severe illness accompanied by polyuria and followed by furunculosis (diabetic) (?). So far "typhus Lannois"—i. e. gigantism. At twenty-seven, first changes of acromegaly occurred—bursting headaches, growing acral pains, slight cerebro-spinal-rhinorrhea. At twenty-nine, failing vision (noted while sighting a rifle), and diplopia set in. At thirty-one, features began to get heavy, enlargement of hands and feet, polyuria, asthenia, drowsiness, loss of libido and potentio sexualis, complete blindness (2nd period of growth) "typus Marie." Height now 6 feet 6 inches, weight 269 lbs., blood pressure only 75-100, blood and urine examination negative. X-ray showed greatly enlarged sella turcica and characteristic tufted ends of distal phalanges. There was divergent squint; bilateral primary optic atrophy and superior hemianopsia of left eye; lymphoid tissue very much enlarged; spacing of teeth; large supple joints; skin moist, smooth and elastic; practically no beard; scant pubic growth of feminine distribution; discrete fatty tumor left shoulder (suggesting Dercum's disease); (in one of Dercum's cases, McCarthy[9] 1902, found an adeno-carcinoma of the pituitary body with testicular hypoplasia); high sugar tolerance (over 300 gms. glucose); temperature subnormal 97-98; testes soft, atrophied; thyroid small, soft. Thymus showed substernal dullness. Adrenal involvement indicated by pigmented skin, asthenia and low blood pressure. Sellar Decompression December 17, 1910, by Dr. Cushing showed chromophobe strumma:—vision temporally improved and headaches stopped. Then recurrence, 1914. Second operation 1914—similar findings—patient got weaker and died. Autopsy February 17, 1915, revealed involvement of most of the endocrinous glands as follows:

Lymphatics of lungs markedly injected.
Thymus—fatty infiltration.
Thyroid—cystic, colloidal goitre.
Spleen—soft, pulpy.
Liver—fatty degeneration.
Adrenals—normal.
Testes—extreme atrophy—tubules contain no spermatogenetic cells—interstitial cells increased.
Lymph nodes: mesenteric, cervical, omental, bronchial, mediastinal } enlarged
Pancreas—interstitial changes.

Tumor—chromophobe adenoma of pituitary body invading temporal lobes.

Hypopituitary states became recognized some fifteen years later than acromegaly. In 1901 Frölich[13] first described the syndrome of adiposity and genital aplasia, with disturbances in menstruation and libido sexualis associated with tumor of the hypophysis cerebri. This condition was named by Bartels[1] 1908 "dystrophia adiposo-genitalis." If the pituitary insufficiency antedates puberty, the secondary sexual characteristics fail to develop and infantilism results. In the male, the body is of the feminine type, breasts hypertrophied, pelvis broad, beard and pelvic hair scanty and of the feminine distribution, etc. If sexual development is complete before pituitary insufficiency occurs, then follow regressive sexual changes, i. e., loss of hair of axillae, eyebrows and pubes; loss of potentio and libido sexualis in the male and amenorrhoea in the female.

Biedl [3] considers these genital hypoplasias as due to changes in the pars intermedia. Goetsch [14] states "both experimentally and clinically there is direct interrelationship between disturbances in the genito-urinary sphere and affectations of the pituitary body. The normal hypophysis seems without doubt to exert a stimulating influence on the development and activity of the genital organs. Excessive activity of the gland is followed by premature development and overactivity of the genital organs: its deficient function results in genital underdevelopment and the non-appearance of sex characters."

Hypopituitarism may be associated with a tumor of the pituitary body proper in which cases we have a primary optic atrophy, and in typical cases a bitemporal hemianopsia; or it may be associated with neighborhood tumors elsewhere in the cranial cavity, which cause a diminution of the pituitary gland function by pressure. In this second class of cases, we have the cardinal symptoms of a brain tumor with choked disc and failing vision, headache, nausea and vomiting, and added to that the regressive sexual changes so characteristic of hypopituitarism.

The following case, recently seen by the author, is cited as representative of this class. The patient, a female stenographer, single, age twenty-six, dates her present trouble from November, 1914, when she began to have backache and pain in the back of the neck. In December, 1914, she collapsed and remained in a state of semi-coma until May, 1915. Since May, 1915, when she gradually roused from her semi-conscious state, she has complained of bi-temporal and occipital headaches, nausea, vomiting, diplopia, failing vision (no hemianopsia), constipation and increase in weight. She began to menstruate at fourteen and flowed regularly for three years. Menses ceased at seventeen and have not since recurred. Examination revealed an obese, lethargic girl of twenty-six with marked old choked disc and nearly blind, (light perceptions in left eye); venules of eyelids dilated, (right external rectus palsy); skin soft, elastic; absence of all axillary and pubic hair; nails dry and brittle; no lunulae; skeleton small; visceral examination normal; temperature 98, blood pressure 108-110; blood Wassermann-negative. X-ray showed normal sized sella with slight thinning of posterior clinoids. Diagnosis of interpeduncular brain tumor, probably from congenital infundibular anlage. Anterior lobe feeding and right subtemporal decompression advised and refused. The regressive sexual changes here are believed to be due to an under-functioning of the pituitary gland which in turn is occasioned by a neighborhood brain tumor pressing upon it.

The author would especially emphasize the complex nature of both cases cited in this paper. The acromegalic after passing through the stage of gigantism ("typus Lannois"), and acromegaly ("typus Marie"), became definitely a victim of an insufficient though greatly hypertrophied hypophyseal gland; as evidenced by the secondary period of asthenia, sexual impotence, obesity, hypotrichosis, etc. During life, he passed through all the phases of hyper- and hypo-pituitarism. At one time, he needed partial hypophysectomy and again actual glandular feeding.

The second case is complex because the pituitary gland is not primarily involved but is the victim of outside pressure influences, decreasing its functionary power. Clinically, it gives the picture of hypo-pituitarism.

Post-mortem examinations in cases of pituitary disease have shown repeatedly—aside from the primary hypophyseal lesion—pathological changes in the other ductless glands; a thyroid enlarged or atrophic, pancreatic islands infiltrated with fat, trophic changes in the adrenals, genital aplasia and even fatty degeneration of the liver. Such findings can almost invariably find interpretation in the clinical history—and the relative hypophyseal influence estimated—for in every case this influence represents but one phase—dominant or recessive—of a general pluriglandular syndrome.

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VACCINE IN TYPHOID.

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In reply to a letter from our secretary I wrote Dr. Jones that I would prepare and read an article on the use of vaccines. I had in mind going over my records and touching the high points in their use in typhoid, gonnorrhœa, whooping-cough, respiratory infections, etc. On looking over my case records and finding that my results with typhoid vaccine differed from the generally-accepted view of little or no effect on temperature, I decided to give only my results with typhoid vaccine. By far my best results have been in tuberculosis, where they have always been uniformly good, more than that, splendid. I am treating at the present time a case of tuberculosis of the larynx. This case came to me from Oakland last March. When she first came you could not hear her speak across my office. Swallowing was very painful. To-day, after three and one-half months' treatment with tuberculin, she is only slightly hoarse, can even sing, absolutely no pain on swallowing, and she has gained some in weight.

In 1908 an article of mine appeared in the New York Medical Journal noting the results I had obtained with diphtheria antitoxin in bronchial asthma. The conclusions I drew were not in entire accord with the conclusions of others at that time, but I believe they are regarded as correct now. While the conclusions I draw in regard to typhoid vaccine may not be correct they may prove of value in some way.

The value of prophylactic typhoid vaccine is beyond question and will not be discussed in this paper. This applies only to the therapeutic use. In Watters' analysis of more than a thousand cases treated with vaccine, they agree in the following results: Patients show less depression, a lower temperature curve, fewer complications, and were brighter throughout the course of the disease.

Their least important effects seemed to be on the temperature curve. While less depression and a brightening-up of the patients were the two points most marked in my cases I did also observe a decided effect on the temperature in the cases where any effect at all was noticeable.

We have been assured over and over again that we can expect no results from therapeutic vaccine because, they say, the symptoms of typhoid are caused by a poisonous split protein from disintegration. Therefore, if we give the protein vaccine we are simply increasing the poison and aggravating the disease so we are not justified in making clinical trial of the therapeutic vaccine. That is theory and test-tube science which does not always apply to the human body, though some of our so-called authorities would like to have us believe it does. Pure physical science works wonderfully in diagnosis, but when it comes to treatment the human body often refuses to become a test tube.

One of the greatest compliments I ever heard given the country-town doctor was given several years ago in New York city. A leader of the medical profession in that city said: "If I were taken very ill I would want Dr. Osler of Baltimore to diagnose my case but I would want a certain doctor up here in a country town whom I know to treat me. In fact I know a large number of country-town doctors, any one of whom I would choose to treat me in preference to our large-town theorists." It is true that this was said in 1902 when drug nihilism was at its height, but it is true to-day in that bedside results are superior to test-tube results, though bedside results are more difficult to obtain and both are essential to best results.

I have used typhoid vaccine in fifteen cases but I am only going to report to you six cases, as they all fall into two divisions and are simply repetitions, cases in which the vaccines gave results and cases in which no effects could be noticed.

Case No. 1. G. R., a boy aged three years. Had been ill two weeks before I was called. He was delirious at first visit. Pulse 150. Temperature 105. Widal was positive. As the patient lived fifteen miles from my office and I had no typhoid vaccine on hand it was two days after I first saw case until I was able to get Widal and the vaccine. During this time temperature continued between 104½ and 105½. Pulse from 150 to 160. Forty-eight hours after first visit I gave this three-year-old boy ½ c.c. typhoid vaccine containing 250,000,000 bacteria. Twenty-four hours later delirium had ceased. Pulse 130; temperature 103½. Forty-eight hours after dose, no delirium, temperature 101, pulse 120. Boy seemed greatly improved and much brighter. The next day he was about the same. The next day he was delirious again and continued so with a pulse around 150 and temperature around 105 for ten days. He received three additional doses of vaccine. He recovered in the usual way of a severe typhoid, the first dose of vaccine apparently giving remarkably satisfactory results with no results whatever from the following doses.

Case No. 2. Sister of above boy, two years old. Began showing signs of typhoid when I first saw case No. 1. She was not very ill but Widal was positive. Temperature 102, pulse 120. I gave her one-half c.c. or 250,000,000 bacteria. Within forty-eight hours temperature and pulse were normal and remained so. The remainder of this family, consisting of an older sister, a two-months-old baby brother, a father and mother, all were given prophylactic vaccine. None contracted the disease.

Case No. 3. R. P., boy aged two and one-half years. He had been ill about one week when I first saw him. Temperature 102, pulse 130. Widal negative. I made a clinical diagnosis of typhoid and instituted typhoid treatment. He gradually grew worse. Widal was made every day. On the twelfth day of the disease it was positive. I now gave one-half c.c. vaccine, pulse being 130, temp. 104. The next day pulse the same, temp. slightly higher. The next day, about the same; I gave a second one-half c.c. vaccine. The next morning temperature fell but rose to 104 during the day. The next day temperature was 101 and the next

day normal, and continued from normal to 100 for three days, then it rose to 102 and I gave another one-half c.c. vaccine. The next day it rose to 103 and the next day fell to 97½ and continued about normal and ended the course of the disease.

Case No. 4. Geo. Witt Sims, aged 5 years. He had been ill ten days when first seen. Temperature 104, pulse 126. Widal negative. Clinical diagnosis typhoid and treatment for same instituted. Two days later, or the 13th day of the disease, Widal positive with temperature 105½, pulse 130. Gave one-half c.c. or two hundred fifty million bacteria. The next day temperature and pulse were practically the same. The second day following vaccine temperature 102, pulse 130. The third day temperature 101½, pulse 110. General condition of patient greatly improved. Temperature continued from 100 to 101 for two days, then began to rise, reaching 103. I gave one-half c.c. vaccine and temperature was 103 the next day. I gave another one-half c.c. vaccine. The next day it dropped to 99½ and never went above 100½ after that, the disease continuing for twenty-four days from the start or eleven after the first dose of vaccine. From the second day following the first dose of vaccine patient was remarkably bright and in splendid general condition.

Case No. 5. F. Ray, girl, aged 4 years. Had been ill ten days when first seen. Temperature 104, pulse 130. Did not make positive clinical diagnosis as it was an atypical case. On the eleventh day Widal was positive. One-half c.c. vaccine given. The next day temperature was 100 and the next day normal, and she had no more fever, temperature above normal and seemed well from then on, or from the third day following the vaccine. This was probably what has been termed an abortive typhoid. We have all known physicians who claimed to have aborted typhoid with calomel or some other simple remedy that most patients get, but which do not stop the disease, and we of course believe this to be a false impression. I am not prepared to say that I think the vaccine cut the course of this case so short. It was probably a case where only a few glands were infected or possibly the intestinal glands were not infected at all but some other organ of the body was the part suffering, though I was unable to prove that to be the case. I assume it was.

Case No. 6. G. M., boy aged 4 years. I saw this case first on the ninth day of the disease. Temperature 103, pulse 120. Widal was negative until the eleventh day, when it was positive with temperature of 105 and pulse of 130. I gave one-half c.c. vaccine on the 12th day of the disease and repeated the dose on the 14th, 16th, and 19th and 22nd days of the disease with absolutely no effect that could be detected. Temperature and pulse ran typical of the severe enteric. Toxemic symptoms severe throughout the disease. Temperature becoming normal on the 27th day of the disease. I have treated two other cases which would be a practical repetition of the above. The cases were treated in 1911 and it is possible that the vaccine was not equal to what it is to-day, or what to my mind is more likely the infection was in each of those cases complicated with the paratyphoid A and B.

One thing noticeable about these cases which I selected to report is the high temperatures early in the course of the disease. I used great care in trying to determine the onset of the disease and believe the days of the disease are correct as given.

I regret that in the above series of cases I am unable to give blood pressure before and after the use of vaccine. I am satisfied, however, that in those cases where a lowering of the temperature was noted there was an improvement in blood pressure, because the most notable improvements were in a brightening of the patient and lessening of all toxic symptoms. I noticed particularly that I began to get results in about thirty-six hours after the injection of the vaccine and that results were marked within forty-eight hours. Unfortunately all the cases in which I have used the vaccine and kept close records have been children. I am unable to report cases of adults. It should also be remembered that no matter how positive the clinical diagnosis I never gave the vaccine until the Widal was positive.

We may sum up as follows:

1. Cases all children.
2. Vaccine never given until Widal was positive.
3. Dose in each case 250,000,000.
4. Effects plainly noticeable in forty-eight hours.
5. No bad effects.
6. Temperature and pulse lowered.
7. Less depression, patient markedly brighter, no hæmorrhage or other complications in any of the cases in which I have used it.
8. Shortening of course of disease possible though I am inclined to believe it only lessens its severity.

The time should soon come when typhoid vaccination shall be compulsory in all schools and places where numbers of people are employed. Then will be the end of typhoid fever.

I cannot close this paper without saying something about diet in typhoid, because in the Medical Clinics of Chicago for November, 1915, which probably many of you read, appeared an article by Dr. Williamson of Chicago. I believe it to be a dangerous article to inexperienced physicians. Here is what he says to feed your typhoid patients during the course of the fever:

Milk, if they like it; eggs, raw, beaten in milk, soft boiled, poached, coddled or omelet; ice cream; cream of wheat; oatmeal or any soft cereal with plenty of cream; soups, not thin "slops" but thick, heavy vegetable soups, as, cream of pea, potato puree, mashed creamed potato; wine jelly, tapioca with fruits; green asparagus tips; cauliflower tips; and he sees no objection to giving sweetbreads and calf's brains.

If he uses that diet there is one thing this man is going to get. That is hæmorrhages. He cannot escape them. He says he has been called out several times to cases of hæmorrhage. We would know that if he had not mentioned it, but he regards it a greater disgrace to have his patients lose twenty-five pounds weight. I have not the slightest fear when I see my typhoid patient lose a little flesh if other conditions are satisfactory, but I have a great horror of hæmorrhage. I have been in two endemics of typhoid and have treated seventy cases. I have been fortunate enough to have hæmorrhage in only one case and that was the case of a man to whom I was called first on

the twenty-first day of the disease, he having been under the care of another physician allowing liberal diet. The other physician was discharged and I was called to the case at one o'clock p. m. I told his wife that he was then bleeding to death. I gave a hypodermic of ergot and morphine and returned to my office for my saline apparatus. When I returned in about one-half hour the bed was full of blood and the mattress was soaked so it ran through on the floor. He died in about three minutes after I reached the house the second time.

I think it was during the years 1901 and 1902 that Dr. H. L. Elsner, physician-in-chief of St. Joseph's Hospital at Syracuse, New York, put every second case of typhoid coming into the hospital on straight milk diet. He put every second one on milk, custard, and strained tapioca. Hæmorrhages were about four to one in those having the more liberal diet. Mortality was also higher but I do not know figures. I believe liberal diet very dangerous except possibly in the hands of the most skillful, and then it is certainly not of enough extra value to equal the extra danger it entails. They tell us there is excessive nitrogen excretion in typhoid so we must feed proteids and immediately say vaccine is of no value because it adds proteids.

My patients get only milk, one tablespoonful of ice cream twice a day and one tablespoonful of gelatine twice a day. I have given liquid peptonoids but failed to see any value from their use and thought my patients showed greater toxemic effects during their use.

Since reporting the above cases I have been treating another case that is interesting in that I did not use typhoid vaccine but Mulford's mixed staphylo-strepto serobacterin.

This patient, a girl aged fourteen years, was first seen on the tenth or possibly twelfth day of the disease. Her temperature was 105 and her pulse 130. I gave one-half c.c. staphylo-strepto serobacterin. The next day temperature was 103½, pulse 130. I gave a second one-half c.c. staphylo-strepto serobacterin. The next day temperature was 101, pulse 126, and all toxic symptoms markedly less. The temperature continued between 100 and 101 for three days when I gave another one-half c.c. of the serobacterin. The next day temperature rose to 102, fell the next day to 100, rose the next day to 102, fell the next day to 100 1-5, the next day to 99 4-5, the next day to normal and never rose above 99 after that. Her pulse and general condition improved with or rather a little ahead of her temperature. This patient was kept strictly on the diet of milk, ice cream, and gelatine, and on August 20th, or thirty days after I first saw her, she had lost only seven pounds in weight. She had no headache after the first dose of serobacterin.

My reason for using serobacterin in this case was that when I first saw her I knew I was dealing with an infection, but it was not at all clear that it was typhoid. By the time I received report on Widal the effect had proven so valuable I continued it.

THE GENERAL PRACTITIONER AND THE TUBERCULOUS PATIENT.*

By ROBERT A. PEERS, M. D., Colfax.

Mr. President and members of the Fresno County Medical Society. Your secretary, in his letter of invitation, requested me to speak to you upon either early diagnosis or the necessity of persistency in treatment when caring for the tuberculous. I thought it would perhaps be better to deal not only with these but also with various other of the problems confronting the general practitioner in the treatment of tuberculosis. Because pulmonary tuberculosis is the form of tuberculosis most frequently met, my remarks will deal exclusively with this type of disease.

The patient with tuberculosis who comes to the specialist for confirmation of diagnosis or for treatment, nearly always comes referred by the general practitioner. He does not apply to the specialist first. If the general practitioner sees the patient early and is keen and capable, the patient's chances for improvement are much enhanced and his stay at the institution shortened materially. If the patient seeks advice late, or if, from faulty diagnosis the nature of his disease is overlooked, the reverse is true. These remarks hold good for the larger number of tuberculous who will not, or cannot, secure the advantage of institutional care. Thus the great burden of correct diagnosis and the care of the tuberculous falls upon the general practitioner and not upon the specialist. Because of this, it is imperative that we should, from time to time, review our knowledge of tuberculosis and endeavor to increase to the utmost our powers of diagnosis and improve our methods of treatment.

The lot of the general practitioner is not an easy one and in his endeavors to secure an early diagnosis and to institute treatment as soon as possible he encounters difficulties other than those which are purely scientific. He is a busy man with but little spare time at his disposal and in the making of an early diagnosis plenty of time is one of the great essentials. Again, he is poorly paid for his time. Only too often there is a "flat rate" fee for office consultations and visits and the patient or his family not infrequently object to paying a sufficient remuneration for the extra time which must be taken. They have come for a tonic or a cough medicine for a patient who is, apparently, merely run down and not only often fail to appreciate the painstaking efforts of the conscientious physician to discover the cause of the "run down" condition or of the cough, but even endeavor to discourage the thought that these symptoms can really be the danger signals of a serious constitutional disorder. Even with the diagnosis of tuberculosis made and the plan of treatment outlined, the physician has difficulty in overcoming the skeptical attitude of his client or the client's family.

But the physician's troubles do not end with the patient and his family. Only too often, it may be

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said to our sorrow and chagrin, he finds opposition from his professional brethren. The patient, not satisfied with the diagnosis, wishing to secure other evidence and anxious to be told there is nothing serious the matter with him, consults another physician. How quickly he believes the other physician who tells him he has merely some trouble in his throat or a bronchitis and "pooh-pooh's" the idea of one so strong looking having any signs of tuberculosis. Such an occurrence is, unfortunately, not rare, and if the patient recovers his former healthy state the conscientious, careful, painstaking physician suffers accordingly. If all such patients recovered, no damage would be done save to the feelings of the careful physician. The reverse is only too true and the patient's "run down" condition becomes worse and sooner or later the diagnosis of tuberculosis is confirmed, perhaps too late to be of benefit to the patient.

I mention these things in passing, because the specialist who sees the patient coming to him late in the disease and with hope for improvement very slim or gone, is inclined, only too frequently, to lay all the blame for the delayed application for treatment at the door of the general practitioner. I mention them also in order to say a word to you to help sustain your faith. These difficulties will arise, they will call forth all your skill, they will cause many a heartache and many bitter thoughts, but, in spite of them, you must be true to yourselves, to your profession and to your clients. They must not cause you to hasten your examination, to underestimate the meaning of the warning symptoms nor to minimize the danger or sidestep the issue when once a diagnosis of tuberculosis is made. Right here I must emphasize the necessity of telling the patient himself that he has tuberculosis. It is not sufficient to tell merely the members of his family. The treatment of tuberculosis requires the active and whole-souled cooperation of the patient, and this can be secured only when the patient realizes exactly the disease he is to fight. If told carefully and tactfully, the diagnosis will not alarm the patient but will stimulate him to assist in the effort made to effect a cure.

It is so easy, when even you may have doubts, and such a great relief to the patient and his family, to assure them that there is no cause for alarm. It saves so much time to write a prescription for a tonic or a cough medicine and is so difficult and time-consuming to order an entire change in the patient's mode of living and outline a regime which will tax the patience and often the faith of the physician, the client and the family. But the tubercle bacillus takes no account of feelings, sympathies or sensibilities. It cannot be bought off by kind words or tender thoughts but can be conquered only by skill, patience and trials.

It is, then, with the idea of reviewing those things which you should know about tuberculosis and to encourage you in the good work you are doing, rather than to attempt to bring before you anything that is new, that I present this paper for your consideration this evening.

In the first place, I would call your attention to some of the more important things in the way of diagnosis, without going into too much detail.

I think that the first mistake that is made in attempting to arrive at a diagnosis is a failure to allow sufficient time for the eliciting of material facts and for examination. Any physician with a proper training and with ordinary diagnostic skill can make a fairly early diagnosis of tuberculosis, if he is in possession of all the facts which should be at his command. He cannot secure these facts relating to history, symptomatology, physical and laboratory findings, without the exercise of patience and tact and the consuming of considerable time. There is no valid excuse for not giving the amount of time necessary. A patient suffering from tuberculosis in a relatively early stage, is not an emergency case demanding immediate attention and if he applies for consultation when the physician's time is taxed to the utmost, he can be given an appointment for some date in the near future when sufficient time can be taken. For this extra time he should pay an extra fee, but whether he pays an extra fee or not, he is entitled to a thorough consideration of his case, or he should be referred to someone who can spare the time. I have given considerable space to what, to some, may seem a very minor detail, because I believe more failures to make proper diagnosis may be traced to haste than to lack of knowledge or skill.

Having made the appointment for examination, a very important aid to diagnosis is a carefully taken and properly recorded history. Such a history will furnish information as to occupations which are such as to lower vitality and predispose to the development of tuberculosis because of long hours, insufficient time for food and recreation, bad hygienic surroundings and faulty modes of living. It will elicit opportunities for infection by the discovery of other cases of tuberculosis with which the patient has come in close contact in the home or otherwise. It will lay bare the patient's medical past and point to previous illnesses which may have been manifestations of tuberculosis unrecognized at the time of illness and which were warning signals of the breakdown which was to follow. A good history will also include a thorough investigation of the train of symptoms which is the incentive that has caused the patient to apply for medical assistance. Upon the facts elicited regarding the past history and the consideration of the present symptomatology may rest the making of a diagnosis much more than upon the findings of the physical examination. For this reason it may be well to state briefly a few of the more important danger signals and symptoms which may help us in arriving at a positive diagnosis.

First as to the danger signals or the past illnesses which are suspicious. We, at Colfax, always consider a history of pleurisy as a pretty positive proof of a tuberculous infection, especially when accompanied by a serous effusion. Likewise a history of malaria is regarded with suspicion if by malaria is meant an illness without frank chills and fever. We immediately inquire if the duration of

the disease was prolonged and if there was an accompanying cough and if the patient lost much weight. We are doubly suspicious if the patient was cured, not by medicine, but as the result of a vacation at the seaside or in the mountains. It has been our experience that more cases of tuberculosis have been wrongly diagnosed as malaria than as any other disease. A history of typhoid or pneumonia is also inquired into and it is remarkable how many so-called cases of typhoid are accompanied by an irregular fever and a cough and night sweats; also how many pneumonias are reported to have dragged over several months. We likewise get the average, the maximum and minimum weights, and, by questioning, learn if the patient is a chronic underweight or if the minimum weight corresponded in time with one of the suspicious illnesses or during a period when the patient was run down and had a cough. Likewise, we ask regarding "clearing of throat" as it has been our experience that most of the cases of definite tuberculosis which we see give a history of an habitual clearing of the throat long before other symptoms arise. This does not, by any means, cover the ground of a well-taken history, but merely indicates a few of the so-called danger signals which are aids to diagnosis.

Now, as to symptomatology. One of the earliest symptoms is a tired feeling—a tendency to get tired after the performance of efforts which once were easily accomplished. This is not always present because active tuberculosis seems at times to occur suddenly and acutely, but, as a rule, the evidence of infection is quite early shown by this tendency to tire easily. It is common in all infections but in acute diseases the other symptoms follow more rapidly than in tuberculosis.

With the tired feeling there is often a loss of weight, but it is really surprising how apparently well nourished a patient may be and how little weight he may lose and yet have tuberculosis advanced far beyond what we would classify as early. But loss of weight is an important symptom and must be given much consideration.

An irregular temperature, a temperature constantly sub-normal, or a temperature above normal, only a few tenths, maybe, especially in the afternoon, are but other evidences of the toxemia of tuberculous infection. Shortness of breath may or may not be present.

Cough and expectoration are later symptoms, as a rule, but are highly significant. Cough and expectoration may be most pronounced in the morning or after eating. It often requires close questioning to elicit the presence of these symptoms. Many patients who cough a few times a day, or even many times a day, will deny having a cough because the cough is not violent and harassing. A slight hack several times a day is not considered by them to be a cough, but must be so considered by us. Women, and sometimes men, often swallow their sputum and will innocently declare that they "never raise anything." Often they claim what they raise comes merely from the nasopharynx.

It is not the history alone or any one of the symptoms alone, but it is a combination of history and symptomatology or a combination of symptoms, which help in diagnosis. It is in the interpretation of the relative value of the facts we elicit in the taking of history and in the compilation of symptoms that experience and judgment find their expression. There may be some excuse for lack of experience or judgment if we fail to make the proper diagnosis, when once the facts are before us, but there can be no excuse for not making the effort to collect the facts.

I shall not attempt to go into the details of the physical examination. I shall merely enunciate a few general principles.

1. Take plenty of time for examination and go over the patient thoroughly.

2. Have the patient in a good light.

3. Have the patient, man or woman, stripped to the waist. No thorough examination of the chest can be made otherwise.

4. In percussion, use only light percussion.

5. In auscultation go over the chest thoroughly. Never consider auscultation complete until you have caused the patient to cough preceding each inspiration.

6. Examine as carefully the chests of supposedly healthy persons who come under your observation as you do those of sick persons. It will give you perspective.

7. Do not expect many physical signs in the early tuberculous. If you find them the case is not early; it is advanced.

Now a few rules as to laboratory, the X-ray and tuberculin:

1. Always examine the sputum. If negative examine again. Always get a morning specimen and instruct the patient to bring it from "deep down." Endeavor to get a positive specimen if possible, but do not allow your laboratory report to outweigh your other evidence as shown by the history, the symptoms and the physical examination.

2. The X-ray is a valuable aid but its findings should be interpreted by an expert. The evidence discovered by the X-ray examination will be of value in proportion to the experience and wisdom of the interpreter.

3. Tuberculin is also an aid to diagnosis, particularly in the young, but, like the sputum examination, must not be allowed to outweigh other evidence. A sputum examination is of value principally when the report is positive; a tuberculin test is of value principally when it is repeatedly negative. Tuberculin, to be of value for diagnostic purposes, should be freshly diluted.

In addition to diagnosis, I should like to touch upon what is, to me, the most striking clinical feature of tuberculosis. I think that too much emphasis cannot be given this particular phase of our subject, because, unless it is fully appreciated, it is not possible to treat tuberculous patients successfully. I refer to the tendency of the tuber-

culous to remissions and exacerbations. All patients exhibit, during the course of the disease, these alternating periods of improvement and periods marked by increase of symptoms. The "danger signals," so-called, already referred to, are usually exacerbations and the long intervening periods of well-being merely remissions.

After the disease becomes definitely recognized as tuberculosis, the "good spells" and "bad spells" continue as before, with the exception that they are now of shorter duration with a tendency towards exacerbation more than remission. As the patient overcomes his disease and progresses toward an arrest of his tuberculous process, the reverse is the rule and the good spells lengthen and become more marked, while the bad spells become fewer and less severe. Until the patient comes to realize that he is suffering from tuberculosis and that the bad spells from which he suffers are but exacerbations of his disease, he will refer to the occurrence of an increase of symptoms as due to "catching cold" a "bilious attack," an "attack of malaria" and so on, ascribing his condition to catching cold if the increase of symptoms is marked particularly by extra cough and expectoration, or to biliousness, if there are gastro-intestinal symptoms or to malaria if the most marked symptoms are malaise accompanied by a rise of temperature.

As I stated above, this clinical feature of tuberculosis must be thoroughly appreciated if we are to treat the tuberculous patient successfully. Otherwise, there will be exhibited by the patient and his physician, an unwarranted optimism when the cough diminishes, the temperature drops and other signs of improvement appear, while there will be a corresponding pessimism when the opposite occurs. Again, as the patient gradually secures an arrest of his disease, when for weeks he may have had a normal temperature and be up to normal weight, there is liable to be a too early return to work or too great liberty allowed in the patient's recreations, unless such good symptoms are treated merely as remissions until all danger of an exacerbation is past. It is a lack of knowledge of these facts or a failure to appreciate their full meaning, which is responsible, usually, for the failure to keep up treatment long enough to secure permanent good results with tuberculous patients.

Various theories have been advanced to explain these phenomena, but probably none is satisfactory. Perhaps none is needed, if we have the patience and tact to insist upon eternal vigilance and unrelenting observance of the rules of hygiene, while at the same time we possess the judgment to interpret at their proper values the significance of these recurring tides of symptoms, which alternately buoy up our hopes or depress us.

Before closing, I wish also to touch on treatment. I am not going to outline for you in detail the treatment of the tuberculous. I wish to speak merely of a few of the more important phases of treatment which I think may be reviewed with value. There are two points where the general practitioner most frequently falls down when it comes to treatment. The first is he does not lay sufficient emphasis upon rest, and the second is he gives tuberculin in too large doses.

First as to rest: The most important single thing in the treatment of tuberculosis during the active stage, is rest. Patients with fever should be placed in bed and kept there until the temperature is normal all day and every day. This means that the temperature must come down below 99° and stay there. This means weeks, and frequently months, spent in bed before the temperature drops to normal. It should make no difference in your treatment how long it takes because your febrile tuberculous patient will not get well unless he is sent to bed and kept there. This seems to be the main fact regarding treatment not yet appreciated by the general practitioner. Because of this lack of appreciation, we are seeing patients daily who have been running a temperature for weeks or months and who have not been put to bed or who have been kept in bed merely in the afternoons. Only rarely do we encounter such a patient who has been advised to exercise. A few physicians apparently have not yet learned that exercise is bad for the febrile tuberculous patient, but they are becoming more scarce day by day. But there are still many who have failed as yet to learn the lesson of rest in bed. Put your patient in bed, serve his meals in bed and do not let him up until his temperature has been normal for days. This is a difficult thing to do in the home; it is fairly easy to do in an institution because the patient can see the beneficial effects in others who are recovering because of rest. In the home the patient does not get the encouragement which comes from seeing many other patients doing the same things as he does, and for this reason the patience and tact of the physician, treating the patient in the home, must be greater.

Now as to tuberculin. Tuberculin is a valuable aid to treatment but when used by the average physician it is given in too large doses. This is due, largely, I think, to the custom of using the commercial dilutions, commencing with dilution No. 1 and going up. Dilution No. 1 contains .01 mgm. to the c.c. and as the commencing dose is one-tenth of one c.c., the patient gets .001 mgm. as the initial dose. This is altogether too large a dose to commence with. We commence with .00001 mgm. to .000001 mgm. Another thing to remember is to refrain from using tuberculin if the temperature is 100° F. or over. There is nothing more sad than to have an advanced patient apply for relief and to learn that though under observation by a physician for many months, he has not been put to bed but has been going twice a week to a doctor's office for a "shot" of tuberculin. We use tuberculin in our institution and we use it because we believe it to be valuable, but we use it in afebrile cases. And when it comes to a matter of relative values, I would not exchange a month's rest in bed for all the tuberculin in existence.

As stated above, I am not attempting to outline the treatment of the tuberculous, but merely to speak of a few of the more important points which I think need to be emphasized. This brings us

to the matter of treatment during convalescence. There is no time in the course of the treatment of the tuberculous when more skill and good judgment is required than during convalescence. I would again warn you not to forget the occurrence of remissions and exacerbations. The patient and his physicians must not allow the occurrence of what may be a long remission to deceive them into believing that a cure has been accomplished. Neither should a slight exacerbation cause them to think that all the months of care and patience have been wasted. As the patient gets well the remissions become longer and more marked, while the bad spells diminish in frequency and intensity.

When the temperature has been normal for a week or more, the patient is allowed up an hour a day unless there are contra-indications, such as a too rapid pulse or great emaciation. Gradually the time up is increased, and, after some weeks, exercise, in the form of walking, is allowed. This is increased as the patient's improvement permits. Here, again, judgment and experience are the greatest aids a physician can possess. The observant physician soon learns that no two patients can be treated alike. He also learns that he must see his patient often and over a long period of time in order to correctly judge his capabilities and resistance. We see our patients at least five times per week, and often daily, over a period of months. At each visit the patient's record is carefully studied. It is in this way, and in this way alone, that we determine just what course shall be pursued with each individual patient.

And this brings up again the matter of time. Time is the great healer of the tuberculous. It is a disease not of days and weeks, but of months and years. We have yet no short cut to cure for tuberculosis. The patient who is willing to devote from six to twelve months or more of his life to getting well, can get well, but he who is looking for a quick cure can be sure of one thing, —he will be disappointed. The cost of getting well is often prohibitive and many persons die in our state each year from tuberculosis for no other reason than that they cannot buy health. This should stimulate us to work for state care for the tuberculous so that patients can be furnished time to get well.

After the patient is able to return to work, what shall he do? This is the question often asked the family physician. Shall he return home or shall he stay in the mountains? Shall he return to his former occupation or shall he take up some light out-door work?

We have one answer to the question of where to live after one has become well enough to return to work. That answer is: "It is not so much *where* you live, as *how* you live." For many months a patient must spend the entire time when not working in bed. Always we advise patients to return home immediately the day's work is done. Often we advise him to go to bed and have his evening meal served in bed. If this is not possible he should retire immediately after his evening meal and stay there until time to get up for breakfast. Sundays should become "days of rest" in the full sense of the term. This may seem unnecessarily strict but the value of such advice is borne out by experience. If possible, the patient should sleep out of doors. He should eat plenty of good food and should keep up his weight. In short, he should follow out, as nearly as possible, the careful regime of the earlier days of treatment. He should take his temperature often and should weigh frequently, not from a morbid interest in his disease, but for the same reason that the merchant or the corporation employs accountants to point out any falling off of business. Truly getting well from tuberculosis is a business to be followed out on business principles. It is necessary to take, frequently, stock of one's resources and gains and to be able to know one's limitations and the sources of one's losses. To live right is more important than to have the right location.

Next, what shall be the occupation? Whenever possible, it should be the one to which the patient is most accustomed and the one which he can follow with the least possible strain. Many indoor positions are more suitable than outdoor occupations, because less strenuous and trying. The occupation to which one is accustomed is the one to which he should return unless there is some positive contra-indication such as too long hours and the impossibility of securing sufficient rest. The problem of the mother of a family with many household and domestic cares, is, perhaps, the most trying one. Here it is necessary to secure sufficient help in the home to help lighten the burdens. The head of almost any household can see the value of spending money for help in the house rather than for doctors and institutional care. The position of those unable to pay for such help is truly pitiable.

In closing I shall, merely for sake of emphasis, review some of the points I have tried to bring out in this short and incomplete paper.

1. Give plenty of time for diagnosis.

2. The diagnosis once made should be truthfully and tactfully made known to the patient. In no other way can his complete cooperation be obtained.

3. We must bear in mind the occurrence of remissions and exacerbations during the course of tuberculosis if we hope to treat the tuberculous successfully.

4. The most important factor in treatment of active tuberculosis is rest.

5. In discharging a patient it is more important to know *how* he is going to live than *where* he is going to live.

6. Discharged patients should return to their former occupations unless there are very distinct contra-indications to so doing.

MEDICAL INSPECTION OF PRISONERS AT SAN QUENTIN WITH REPORT OF CASE OF TINEA VERSICOLOR.

By L. L. STANLEY, M. D., San Quentin.

Each prisoner upon entering San Quentin prison is subjected to a thorough physical examination. As soon as he enters he is taken to the turnkey's office, properly registered, and instructed as to his privileges and requirements. After a bath, a shave, and a cropping of the hair he is taken to the Bertillon room, where his physical measurements are tabulated, as well as any scars, deformities, birthmarks and the imprints of the palmar surfaces of his thumbs and fingers. His face is photographed both from in front and laterally for records of identification.

At the hospital, where the prisoner next goes, a complete medical history is taken by one of the physicians who are serving sentences. This history begins with date, age, occupation, weight, family encumbrence, and length of sentence. The family history has to do with the health, drug and alcoholic addictions, sanity, tuberculous and malignant tendencies of both maternal and paternal sides. The health of the brothers and sisters, with notes of family deaths, is carefully recorded.

The past history of the convict embraces childhood diseases, infections, accidents, venereal troubles, stomach and bowel disturbances, nervous peculiarities and all affections of the heart and lungs. Alcoholic, drug and tobacco addictions are written down.

The physical examination is dictated by one of the prison physicians to a scribe who places it on the history blank. The examination starts with the noting of the condition of the head, its scars, deformities and peculiarities. The color and size of the ears and hearing ability are noted. The eye examination includes the color of the conjuctivæ and scleræ, equality of pupils and their reaction to light and accommodation. The air spaces, spurs and septal deflections of the nose are noted. The condition of the lips, teeth, gums, tongue, tonsils and pharynx is carefully inspected.

Glandular enlargements, abnormal pulsations, hyperthyrea and muscular condition of the neck is examined in turn. The chest is inspected, palpated, percussed and auscultated during normal breathing and by holding the breath and coughing. The heart measurements are taken, pulse rate, rythm and murmurs, if any, noted.

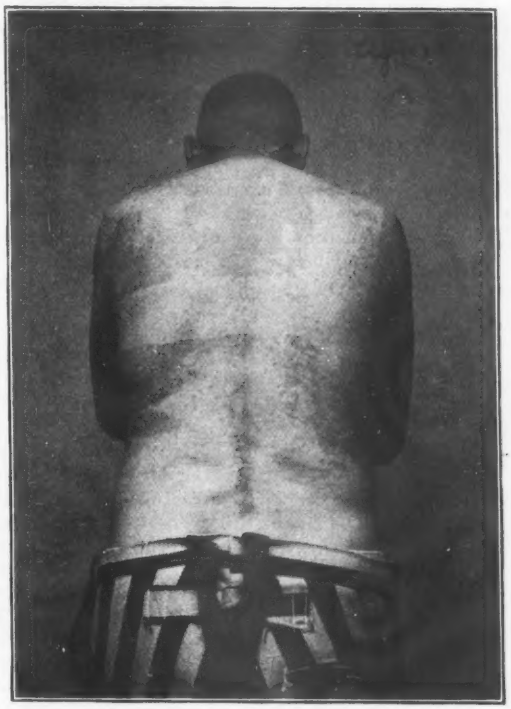
Posteriorly the lungs receive the same examination, stress being laid on the clearness of the bases and apices.

The area of liver dullness and the percussability of the spleen are included in the examination of the abdomen.

The external genitals are carefully examined for acute infections, varicocele, hernias and other affections of this region. Note is made of the musculature of the legs and arms, tendencies to arteriosclerosis and varicosities.

Reflexes and glandular enlargements are ascertained.

During all this examination, if there be any remediable defects, such as enlarged tonsils, deflected septum, hernia, varicocele, etc., such defect is recorded in the "Operation Book," and the prisoner advised that he will be called at an early date for the necessary operation if he so desires.



Upon completion of the examination a signed statement of the prisoner's physical condition is sent to the Captain of the Yard, who has charge of the detailment of the men to their work. Recommendations are made by the physician as to the kind of work the physical condition will allow. No man is assigned to duty in the jute mill who has epileptic fits, tuberculous tendencies, eye affections or physical deformities which contraindicate such work.

When it is realized that approximately three prisoners enter San Quentin prison every day, it is obvious that many interesting cases come before the medical directors.

The accompanying photograph shows some peculiar skin markings of a convict who came to the prison in February, 1913. Upon being stripped, a brownish, somewhat elevated discoloration was noticed covering the upper part of the thorax and the upper part of the abdomen as well as about the genitals. Extending in a band eight c.m. in width across the chest just above the nipples and around on to the back and at right

angle over the right shoulder, simulating Sayre's bandage, was an area almost entirely devoid of this pigmentation and presenting almost entirely normal skin.

Patient's family history not applicable to case—age 50 years, married, real estate broker, weight 194 lbs., previous health good. Gonorrhœa 30 years ago, no syphilis, moderate drinker and smoker. Has been troubled with constipation and hemorrhoids. History otherwise negative.

Present history—Prisoner had never noticed pigmentations of his skin before, and had not had any itching or other trouble to direct his attention to the skin. He was surprised at his markings when attention was attracted to them, but declared he had never worn tight-fitting clothing or been supported by braces. On close questioning, however, he remembered in April, 1912, nine months before, he had dislocated his right shoulder. Adhesive plaster had been applied about his chest, back and shoulders, but on account of its irritability it was removed within forty-eight hours.

For three months the patient had been confined in a county jail before coming to San Quentin.

Physical examination showed patient normal in other respects.

A portion of the brownish skin was scraped off and mounted with 10% sol. of sodium hydroxide.

With 1/6 objective, the mycelia were plainly visible substantiating the diagnosis of Tinea versicolor.

In order to prove the curative power of adhesive plaster in this condition, strips of ordinary plaster were placed over areas where the tinea was thickest. These strips were left on 24 and 48 hours respectively. On removing the first one the skin appeared to be normal and some of the brownish discoloration remained on the plaster. A few patches subsequently reappeared in this area.

The second strip applied for 48 hours has permanently cured the disease over the area on which it was applied.

One area where it was impracticable to apply adhesive a 12% solution of sodium hyposulphite was applied several nights in succession. This succeeded in curing the disease.

A CASE OF HEMOGLOBINURIA.

By NORMAN E. WILLIAMSON, M. D.,

Emergency Hospital, P. P. I. Exposition, San Francisco, California.

The following case of hemoglobinuria presents some points of interest aside from the rarity of the condition here.

E. F. W., age 46, born in Boston, Mass., occupation, guard, P. P. I. Exposition. First seen March 9, 1916. Patient complains of highly colored urine, which he first noticed two days before, accompanied by severe repeated vomiting. Has not been feeling well for two weeks, having had "flashes" of fever, insomnia, and anorexia.

Father died at 46 of tuberculosis; mother at 56 of rheumatism. Two sisters, one of whom died of measles, and the other of childbirth. One brother, alive and well at 54.

In early childhood patient had whooping cough, diphtheria, measles, scarlet fever and mumps. Had epilepsy with attacks every three or four months up to the age of 13, and none since. Pneumonia 20 years ago. Typhoid in 1896. Went south first in the army in 1898, and had repeated attacks of malaria in Florida. Went to Manila in 1899 and had no malaria till 1906, when an attack kept him in the hospital for a month. From that time he had no known attacks of malaria, nor any other illness to the present time, except gonorrhoea in 1911. Never had syphilis. Came to San Francisco from Manila, December, 1914, and has never left the city since, even for a trip. Has taken no medicine.

The patient is a well-built, rugged-looking man, weighing 185 lbs. Face flushed. Sclera faintly yellow. Temperature 99.2°. Pulse 72. Blood pressure, systolic 138, diastolic 85. Chest negative. Spleen not palpable or tender. Liver, normal. Slight tenderness on deep palpation over left kidney and bladder. Urinates frequently, and has slight burning sensation on urination.

Urine. Reaction, strongly acid; specific gravity 1010. Albumin by nitric acid layer test +++. Turbid. Yellowish-red by transmitted light; reddish-brown by reflected light. On standing there is a light brown flocculent sediment. On centrifugation of the fresh specimen there is 3% of brownish-black compact sediment, and a brown clear fluid. Microscopically, the sediment is nearly all amorphous brown material, with a very few red cells, leucocytes and epithelial cells. Negative for B. Tuberculosis. The blood does not show any parasites of malaria or pigment in the fresh spread or in the layer beneath the leukocytes in the centrifugated specimen. (Bass method) 2cc. of blood were added directly to 5cc. of citrated salt solution and centrifugated. The clear supernatant fluid showed a color, as viewed through a test tube, 16mm. in diameter, estimated as 15% by comparison with the Tallquist scale.

The stool was negative for ova of animal parasites. The complement fixation test, performed by Clegg in the Public Health Service Laboratory, was negative, both with Wassermann and Noguchi antigens. The blood for this test was drawn March 13. At the same time 1cc. of blood was added to 5cc. of citrated salt solution and centrifugated. the supernatant fluid was of a pale straw color. No malarial pigment was found on examination of the leucocyte layer. The specimen was shaken and left for 24 hrs. at room temperature. No hemolysis occurred.

The urine improved slowly, changing in color to yellow by March 16. Hemoglobin derivatives were shown by Heller's test till March 25, and by the benzidine test on the next day. Casts of brown amorphous material appeared, and on the 21st there was an increase of red cells. Albumen was found daily by the nitric acid layer test till March 27, when this test was negative.

Blood examination following admission showed: red blood cells 4,000,000. Hemoglobin (Tallquist, 65%. Leucocytes 6000. Slight poikilocytosis with shadows. No nucleated reds.

Polymorphonuclear neutrophiles	63%
Large mononuclear	19%
Lymphocytes	7%
Transitional	3%
Neutrophile myelocytes	4%
Polynuclear eosinophiles	4%

On the next day the hemoglobin was 55%. Two days later the results were as follows: red cells, 4,300,000; hemoglobin 55%.

Polymorphonuclear neutrophiles	62.5%
Large mononuclear	13.5%
Lymphocytes	19.5%
Transitional	1.5%
Neutrophile myelocytes	1.5%
Polynuclear eosinophiles	1.0%
Mast cells	0.5%

On the 23d the hemoglobin was 68%; red cells, 4,600,000. The patient lost 18.5 lbs. during the first 11 days, and gained a half a pound in the next week.

There was no history in this case of the ingestion of any of the various drugs and chemicals which can produce hemolysis. The patient had taken no medicine, and no one living in the same house was ill. He had eaten no mushrooms. There was no history of exposure to cold. Burns, snake bites and Raynaud's disease can be excluded.

Justus showed that hemolysis occurred in certain cases of syphilis, and that the serum might be red in such cases when drawn from a vein and separated. He caused hemolysis in syphilitics by intramuscular or intravenous injections of mercury. Murri showed that 90% of cases of paroxymal hemoglobinuria had syphilitic history, and that the attacks could be induced by a chill. Cooke stated that syphilis is the most important, if not only, cause for paroxymal hemoglobinuria. Syphilis is excluded in this case by the history, physical examination and negative complement fixation tests.

Tropical black water fever remains for consideration. This patient had had a prolonged tropical residence. He had attacks of malaria in 1898 and again in 1906. At the time of examination no malarial parasites could be found, but, according to the history he had passed highly colored urine for 48 hours. Parasites disappear inside of 48 hours in black water fever, as any of us who have had experience with the disease can testify. The infected corpuscles are the first to be broken up, and a patient may be free from malaria for some time after an attack of black water fever. The patient, however, was not pigmented, and showed no splenic enlargement or tenderness. No pigment could be found in the leucocytes. There was a low leucocyte count, 6000, which would correspond to the usual finding in malaria. Hemoglobinuria may be accompanied by leucocytosis.

Rogers first noted the increase of large mononuclears in malaria. Houston stated that large mononuclear increase was present also in splenic enlargement, some acute septic conditions, some cases of chlorosis and the anemia of typhoid. Stevens and Christopher state that an increase of 15% of large mononuclears means actual or recent malarial infection, and that an increase of 20% is accompanied by parasites and pigment in the blood.

Taking 3% to 5% as the normal proportion of large mononuclears there was an increase of 14% in the first count made. This dropped in two more days to an excess of 8½%. In the absence of the other conditions mentioned by Houston, this is strongly suggestive of a recent attack of malaria. The symptoms of malaria, especially of the estivo-autumnal form, may be very slight, and the patient complained of indisposition for two weeks before the attack of hemoglobinuria. Malaria often recurs in the north when it had been latent for some time before leaving the tropics. No new infection could have occurred here, as the patient had not been outside of San Francisco, and Anopheles are not present here.

According to Koch, Plehn and others quinine is a cause of black water fever in infected individuals. According to Stevens and Christopher, five grains of quinine may induce an attack when given to an infected individual. All cases are not so caused, however. I have treated patients with large doses of quinine through repeated severe attacks of malaria, and the same case has subsequently developed black water fever. In many cases quinine given during the disease does not seem to alter its course. Taking of quinine is so nearly universal in the tropics, especially when indisposed, that it is rare to find a patient with black water fever who has not taken quinine. This case had had no quinine.

The urine differed in no way from those seen in many cases of hemoglobinuric fever seen in Panama by the author. I consider that the weight of evidence is in favor of this diagnosis.

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Justus, J.—Virch. Archiv. CXL, 1895 and CXLVIII, 1897.
Murri—quoted by Justus.
Cooke—Amer. Jour. of Med. Sci., Vol. 144, 1912, p. 203.
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Houston—Brit. Med. Jour., Sept., 1904.
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Book Reviews

The Medical Clinics of Chicago. Volume II, Number 6 (May, 1917). Octavo of 252 pages, 46 illustrations. Philadelphia and London: W. B. Saunders Company, 1917. Published bi-monthly. Price per year: Paper, $8.00; cloth, $12.00.

General Surgery. Edited by Albert J. Ochsner. Vol. 2 of Practical Medicine Series for 1917. Pp. 608. Chicago: Yearbook Publishers. 1917. Price $2.00.

Internal Secretions; their physiology and application to pathology. By E. Gley. Translated from the French and edited by Maurice Fishberg. Pp. 241. New York: Paul Hoeber. 1917. Cloth, $2.00.

This little book is an effort to condense the present status of the theory of internal secretions into a small work intended for the busy practitioner. In reading it through, the latter should be prepared for a very general, somewhat advanced presentation of the subject, which presupposes considerable fundamental knowledge. Perhaps its most glaring fault is the almost entire disregard which is paid to the actions of the hypophysis. As a single work on the subject of the internal secretions it is not to be compared with either Biedl or de Sajous. H. F. A.

Mechanisms of Character Formation. An introduction to psycho-analysis. By William A. White. New York: Macmillan. 1916. Price $1.75.

This is a very serious book dealing with the application of the Freudian theories and psychoanalysis to every-day life. Its chapters deal with the genetic approach to the problem of consciousness, symbolism, family romance, the will to power, and many similar abstruse considerations. It is an excellent work for those who are interested in psychic problems, but rather beyond the comprehension of the average doctor. However, a greater familiarity with these phases of medicine would be extremely helpful to the average practitioner. S. T. P.

Diagnosis from Ocular Symptoms. By Matthias Lanckton Foster. New York: Rebman Company. 1917.

This book will prove of interest mainly to the teacher of ophthalmology looking for an outline of important clinical points to present to his students, or to the student himself to supplement literature. The main lesions of the eye and their significance are discussed in simple manner, but with excellent clinical judgment. The unessentials are disregarded. It would be a good book for the general medical man to possess as it gives short and well put clinical facts and discussions of important eye lesions. H. B.

Venesection. Brief summary of the practical value of venesection in disease. By Walton Forest Dutton. Philadelphia: Davis. 1916. Price $2.50.

It is undoubtedly true that venesection is a neglected form of treatment and is applicable in many disease conditions. The past history of this useful procedure has been well written by medical authorities long since dead.

It still remains for some modern author to deal adequately with the topic. The book above mentioned probably is a serious effort to do this, but it fails woefully to come up to any modern scientific standard. It deals with venesection as a treatment to many diseases, such as angina pectoris, beriberi, yellow fever, migraine, etc. But an author who can state that "rheumatism is a constitutional disease, the cause of which has not been determined, and that it may be acute or chronic," is hardly to be taken seriously, even should he recommend venesection. Even the illustrations in the book, which are undoubtedly taken from some antiquated instrument catalog, are more amusing than illuminating. Therefore we can only recommend the book as one to skim over and pick out such suggestions as may stimulate further investigation and more accurate clinical observation.

S. T. P.

Pulmonary Tuberculosis. A handbook for students. By Edward O. Otis. Boston: W. M. Leonard. 1917.

This little book should fill quite a want in our library of tuberculosis. It adds nothing new, but presents the subject in condensed form from the viewpoints of a teacher of many years' experience. The book is rather a compendium on tuberculosis and is written and planned for the medical student primarily.

The chapters upon prognosis, prophylaxis and marriage and tuberculosis deserve special mention. The intelligent co-operation of the patient as affecting his recovery is emphasized, a point much to be commended.

The numerous conditions which produce tuberculosis, beginning with childhood infection; the strengthening of natural resistance in young adult life; the prevention of active tuberculosis from latent infection; the improvement of working and housing conditions, are impressed upon the reader.

Finally, the author does not absolutely forbid marriage between tuberculars, but warns against a woman bearing a child until two years after the disease has been arrested, and then only if her status in life is such that no great demands are made upon her strength. Under these conditions a healthy child may be reared. An arrested tuberculous male may marry if his responsibilities are not too burdensome.

As a practical abridged sketch of the entire subject of pulmonary tuberculosis, this book can be recommended to students of medicine.

W. C. V.

Clinical Tuberculosis. By F. M. Pottenger. With a chaper on laboratory methods by J. E. Pottenger. 2 volumes. St. Louis: Mosby, 1917. Price $12.00.

These two large volumes on tuberculosis view the subject from the standpoint of pathological anatomy, pathological physiology, diagnosis, prognosis, complications and treatment. They may be used for reference on certain subjects of special interest or for more extended reading. Each chapter is very complete in itself, and this, I believe, is one of the strong points in favor of this work compared with many where it is necessary to spend much time referring to other chapters. Each section is conscientiously and fully covered and supplemented by many charts, diagrams and X-ray photographs.

The writer, among other things, has gone considerably into detail in regard to the part played in clinical tuberculosis by the vegetative nervous system. As he admits, that system does not account for all the signs and symptoms, but he has carefully described and elaborated the possibilities.

The pathological anatomy and physiology of the infection is traced from its appearance in childhood though its often latent period to the active disease of adult life. By carefully understanding the transition an early diagnosis is more often made than would otherwise be possible. Considerable attention is paid to these early signs and symptoms and their importance.

The complications of tuberculosis cover a very large field and different methods of treating them is of importance to all of us.

In conclusion, these volumes of Pottenger's, divided as they are into small chapters, make very interesting and instructive reading for reference as well as study and in these 1400 pages all different aspects of the disease are considered and thoroughly discussed. P. H. P.

A Text-Book on the Practice of Gynecology. For Practitioners and Students. By W. Easterly Ashton, M. D., LL. D., Professor of Gynecology in Graduate School of Medicine of the University of Pennsylvania. Sixth Edition, thoroughly revised. Octavo of 1097 pages with 1052 original line drawings. Philadelphia and London: W. B. Saunders Company, 1916. Cloth, $6.50 net; half morocco, $8.00 net.

The sixth edition of this very complete gynecology will be an especially welcome addition to the library of the young practitioner. The changes are many, including not only treatment, conservative and operative, but also bacteriology and pathology. Into a book of 1100 pages is crowded not only the specialty of gynecology, but also chapters on intestinal surgery and kidney surgery. One wonders a little—why not go on to gall-bladder surgery? However, the author probably does attempt to remind one that it is necessary to be a general surgeon before becoming a gynecologist. This must be continually emphasized, for one still sees the abdomen opened for a simple suspension and the gall stones left intact, not even palpated.

The chapter on cancer of the uterus is decisive and the procrastinating medical man is prodded to immediate diagnosis. Description and pictures are so complete that the mind absorbs all the details without effort. The author leaves not the slightest detail to the imagination. This, while a little irksome to the experienced man, is all the more valuable to the young practitioner. In this book he will find many happy suggestions. As in a reference book, each condition being treated in full and systematic fashion, no time is wasted in hunting through other pages. The young doctor beginning to teach will find this book especially excel-

lent, as it emphasizes the small details so valuable to the student and too often taken for granted by the general instructor. Treatment is complete, with chapters on hydrotherapy, X-ray, diet and saline injections. At first glance one misses the halftones of other books, but the drawings prove very satisfactory.

Altogether the book can be well accorded a place in the well-equipped medical library.

M. I. J.

Post-Mortem Examinations. By William S. Wadsworth, M. D., Coroner's Physician of Philadelphia. Octavo volume of 598 pages, with 304 original illustrations. Philadelphia and London: W. B. Saunders Company, 1915. Cloth, $6.00 net; Half Morocco, $7.50 net.

The author, as stated in his preface, has based his book and his illustrations on his personal experience and thought, which covers a period of sixteen years and an observation of more than four thousand post-mortems. He states, at the outset, his purpose to try to encourage a more scientific and thoughtful study of the human body on the part of the post-mortem surgeons, and to stimulate higher ethical standards and a broader view, which is offered those engaged in this profession. The book as a whole is well gotten up and orderly in the arrangement of its contents. The topics are well outlined and indexed. The reading matter, from cover to cover, reveals a passion on the part of the author to remodel or revise some of the present post-mortem methods and customs. His criticisms of the technic and practices of some of the present day post-mortem surgeons is at times severe. Especially does he criticize the careless worker, also those who methodically follow a set routine, particularly the students of Virchow.

However, in spite of tendency to criticise, he, himself, occasionally drops into the straits from which he would warn others; as, for instance, his constant reminder that one should search for facts based on direct evidence rather than on notion or biased statement, seems somewhat vitiated by such statements as appear on page 67, in the section on hair, where the following appears: "The association of red hair of a deep brownish copper type with blood vessel disease is most striking. I have come to speak of this type as hemic hair. The coarse sparse hair of gastritis and the fine buff colored hair of old liver degeneration are very characteristic." Personally I have never seen or heard of such associations, but this may be due to lack of observation on my part.

It seems the book is so full of detail, that it should in a general way belong to an anatomical dissecting room, rather than for use in the average run of post-mortems where the operator's time is quite limited by the various demands, of undertakers, friends and relatives, etc., which interfere with careful lengthy dissections of any but the particular parts involved. The book also carries one afield somewhat into the realms of physics, chemistry, physiology and anatomy, probably more than is indicated under the present title.

In general, there is much that can be obtained by way of the author's personal experience and suggestion that would be helpful to anyone interested in post-mortem work. I consider his chapters on Phenomena of Death (Part 1), Mortuaries (Part 2), Examination of the Body (Part 3), and Medico-Legal Post-Mortems (Part 5), quite interesting, instructive and worth while. E. W. S.

State Society Notes

IMPORTANT NOTICE—TO CONTRIBUTING MEMBERS OF THE INDEMNITY DEFENSE FUND.

Notes are now becoming due.

Do not let your membership lapse.

Each member will be informed ten days in advance of the due date of his note.

DEFENSE AFFORDED ONLY TO MEMBERS WHOSE DUES ARE KEPT FULLY PAID.

Medical Defense Rules, Section 3: "Dues must be paid to the Secretary of the County Medical Society to which each member belongs prior to the end of February of each year. Any member whose dues are not paid prior to March 1st and whose name is not reported as having paid his dues by the Secretary of his County Medical Society is dropped from the list of members in good standing as of January 1st of such year, and such member is deprived of Medical Defense afforded by the State Society for the period from January 1st of such year to the date when his assessment is received by the State Society. Members whose assessments are not received on or before February 15th of each year will be notified by letter from the Secretary of the State Society of such fact."

At a recent meeting of the Council of the Medical Society of the State of California, the question was raised as to what stand the insurance companies had taken on the matter of service in the Medical Section of the Officers' Reserve Corps in the present war. Dr. Pope, State Secretary, is investigating the matter of insurance rates, recent rulings, if any, and what provisions are made for physicians in the present crisis. A resumé of this inquiry will be furnished in the near future.

NOTICE.

Information has come in the possession of the Police Department of San Francisco to the effect that a middle-aged German woman has been going the rounds of the doctors' offices offering for sale a mouthpiece attachment for the telephone, also a memorandum pad to attach to the telephone, both of which are said to be laden with disease germs of some description. Should any physician come in contact with this woman, he should detain her on some pretext and notify the police.

County Societies

CONTRA COSTA COUNTY.

The Contra Costa County Medical Society met in regular monthly meeting at the residence of Dr. Marguerite Deininger-Keser, Saturday night, June 30. The minutes of the previous meeting were read and approved. There was no unfinished business to come before the meeting.

A letter from the Editor of the Journal was read, in which it was stated that an associate editor from the County Society be elected. A motion was regularly moved and seconded that Dr. U. S. Abbott be elected associate editor of the State Journal in Contra Costa County. The motion was carried.

A motion was regularly made and seconded that the President appoint a committee of three members to draft resolutions to protect the doctors' practices while away during the period of the war. The motion was carried. Other communications were read.

Dr. Jas. T. Watkins of San Francisco gave a very interesting talk with lantern slide illustrations on the subject "Injuries and Deformities of the Upper End of the Femur." This was an unusually interesting talk and freely discussed by the members present.

Dr. L. A. Martin of Oakland gave us a very interesting talk on X-ray work.

At the conclusion of the regular program Dr. Keser served a very pretty luncheon, for which she was given a vote of thanks.

Those present were: Dr. P. C. Campbell, Richmond, President of the Society; Dr. H. L. Carpenter, Richmond; Dr. C. R. Blake, Richmond; Dr. W. E. Cunningham, Richmond; Dr. U. S. Abbott, Richmond; Dr. M. Deininger-Keser, Richmond; Dr. W. W. Frazer, Richmond; Dr. H. N. Belgum, Richmond; Dr. C. R. Leech, Walnut Creek, Vice-President of the Society; Dr. G. O'Malley, Crockett; Dr. M. L. Fernandez, Pinole; Dr. W. S. George, Antioch; Dr. Moore, Antioch; Dr. J. T. Breneman, El Cerrito; Dr. F. F. Neff, Concord.

U. S. ABBOTT, Secretary.

SACRAMENTO SOCIETY.

The regular monthly meeting of the Sacramento Society for Medical Improvement was held Tuesday evening, June 19, 1917, at the Hotel Sacramento.

The following program was presented:

Cases reported: Dr. Zimmerman presented X-ray plates of Transposed Heart, and Foreign Body in the Lung. Dr. Gundrum reported a case of Cerebro-Spinal Meningitis, and demonstrated an unusual fever chart for this disease. Dr. Rulison reported a case of Laryngeal Diphtheria, complicated by an Emphysema. Dr. Pitts reported a case of Congenital Heart Disease.

Paper of the evening: Artificial Pneumothorax. Discussion was opened by Dr. Kirkwood of Alta. Discussion continued by Dr. R. A. Peers, Dr. Bush of Colfax, and Dr. Gundrum. Discussion closed by Dr. Howard.

Report of the Board of Directors was read.

Meeting adjourned.

W. A. BEATTIE, Secretary.

SAN FRANCISCO COUNTY.

At a special meeting of the Board of Directors, held on June 28th, the following resolution was adopted:

"Resolved, That the members of the San Francisco County Medical Society as a whole, when called upon to treat persons who are patients of physicians who have given up their practice for the purpose of entering military service during the present war, will render such services as may be required and collect the usual fees, one-third of which amount collected will be immediately forwarded to the family or proper representative of the above mentioned medical officer. Be it further understood that this shall be done whether or not the absent medical officer shall have stipulated to whom the patient shall be referred; be it further

"Resolved, to the end that each member of the San Francisco County Medical Society may have an opportunity of verifying this action, that the Secretary of the Society shall transmit to each member for his signature the following:

"'I hereby subscribe to and agree to abide by the provisions of that Resolution of the Board of Directors of the San Francisco County Medical Society providing that I shall transmit to the family or proper representative one-third of all fees received from patients of medical officers who have given up their practice to enter military service.'"

RENÉ BINE, Secretary.

SAN JOAQUIN COUNTY.

The regular monthly meeting of the San Joaquin County Medical Society was held at the home of Dr. J. P. Sargent at Lodi Friday evening, June 29th. The meeting was called to order by first vice-president R. T. McGurk. Those present were Drs. E. A. Arthur, B. F. Walker, R. T. McGurk, W. T. McNeil, J. T. Davison, Margaret Smyth, Minerva Goodman, Hudson Smythe, S. R. Arthur, J. E. Nelson, A. M. Tower, E. B. Todd, J. P. Sargent and H. J. Bolinger, with Dr. McCloskey and Dr. B. M. Kraut as guests.

The program of the evening was a Symposium on Military Topics. Dr. B. F. Walker addressed the society on his military experience with the French army, explaining the difference between the base hospital, clearing hospital and convalescent hospital. Dr. Hudson Smythe spoke on his anticipation of service with the University of California Red Cross Unit. First Lieutenant B. M. Kraut, U. S. Army, gave a very pleasant talk on emergency treatment of war wounds and fractures, and Dr. J. E. Nelson explained the purpose of the San Joaquin County Red Cross Unit.

At the close of the program, a very delightful buffet luncheon was served by Dr. Sargent.

H. J. BOLINGER, Sec. Pro. Tem.

SISKIYOU COUNTY.

The Siskiyou County Medical Society held its regular quarterly meeting at Shasta Springs, Monday, July 2nd, at 2 P. M.

Dr. Leo Eloesser of San Francisco gave an interesting talk on injuries of the joints as ob-

served by himself while serving as surgeon at a large base hospital in Germany during the present war. Dr. Eloesser's talk was illustrated by lantern slides and he clearly demonstrated how the mortality decreased as the treatment of the injuries improved.

Dr. Theodore Rethers of San Francisco followed with a discussion of the German methods.

Dr. Hal Warren of Montague read an interesting paper on Angina Pectoris and Dr. C. W. Nutting, Jr., of Etna Mills read one on a valuable method of treating inevitable abortion when assistance was not to be had.

At the business meeting Dr. C. W. Nutting, Sr., of Etna Mills was elected President, Dr. A. A. Milliken of Fort Jones, Vice-president, and Dr. H. R. Parker of Dunsmuir, Secretary and Treasurer.

A banquet was served by the Shasta Springs Hotel Co. for the members and their wives at 6 P. M.

H. R. PARKER, Secretary.

EXAMINATION QUESTIONS—STATE BOARD OF MEDICAL EXAMINERS JUNE, 1917.

Surgery.

P. T. PHILLIPS, M. D.

10:00 A. M. to 12 M., June 11, 1917.

1. Discuss briefly enteroclysis, drip method (Murphy), and its uses in surgery.
2. How would you treat a gunshot wound involving the elbow joint?
3. Describe acute suppurative otitis media, giving treatment. Name the complications that may arise if not properly treated.
4. Give the causes and treatment of sympathetic ophthalmia.
5. Describe briefly the different accepted methods of treating burns.
6. Describe in detail the method of using Carrel-Dakin solution in the treatment of infected wounds.
7. Describe dislocations of the astragalus, giving causes and treatment.
8. Describe symptoms of perforated typhoid ulcer and give surgical treatment in detail with prognosis.
9. Describe in detail treatment of fracture of patella.
10. Discuss briefly drainage of the abdominal cavity. (a) When to drain. (b) How to drain.
11. When would you advise nephrectomy? Describe operation in detail.
12. How would you treat a deep infected wound of the palm and what are the dangers of improper treatment?

(Answer ten questions only.)

Physiology.

ERNEST SISSON, D. O.

1:30 P. M. to 3:30 P. M., June 11, 1917.

1. Describe the olfactory stimulus and discuss relations of odors and taste.
2. Explain the variations in pulse rate caused by change in properties or composition of the blood.
3. Describe the nervous mechanism involved in vomiting.
4. Discuss the specific qualities of enzymes.
5. Describe the functioning of the gall bladder and causes of same.
6. How does the venous structure in the brain differ from that in other parts of the body?
7. What is the physiological process of recovery from pneumo-thorax?
8. Discuss the cerebro-spinal fluid as to its formation, location and function.
9. Describe the excretory functions of the skin giving nerve regulation.
10. Describe the relation of the ovaries to menstruation.
11. What effect has peripheral resistance on arterial and venous pressure?
12. What protective reflex would be exercised upon contact with irrespirable gases?

(Answer ten questions only.)

Pathology and Bacteriology.

DAIN L. TASKER, D. O.

4:00 P. M. to 6:00 P. M., June 11, 1917.

1. Discuss normal histolysis and cytolysis.
2. What diseases produce infective granulomas in the testicle?
3. Describe the pathology in acute osteomyelitis.
4. Give the blood picture in pernicious anemia.
5. What is the significance of leucocytosis?
6. Discuss gall stones.

NOTE—In the following six questions the examinee is privileged to select for his discussion a disease which typifies the class of infecting organs noted. Make all discussions brief.

7. Discuss the characteristics of some one member of the micrococcus group of bacteria and describe the body's typical reaction to it.
8. Discuss the characteristics of some one member of the bacillus group of bacteria (spore bearing) and describe any local lesion which is characteristic.
9. Discuss the characteristic pathology of some one member of the group of diseases caused by filterable viruses.
10. Discuss the causative organism and the pathology which characterizes some one disease among those due to yeasts.
11. Discuss the causative organism and pathology which characterizes some one disease due to a protozoan infection.
12. Discuss the causative organism and the pathology which characterizes some one disease due to a metazoan parasite.

(Answer ten questions only.)

General Medicine Including Clinical Microscopy.

For Physician and Surgeon Applicants.

H. L. ALDERSON, M. D.

June 12, 1917.

1. Discuss the symptoms, and prophylactic and active treatment of chlorine gas poisoning.
2. Discuss the etiology, diagnosis and treatment of amoebic dysentery.
3. Discuss briefly the symptoms and diagnosis of hyperchlorhydria.
4. Discuss briefly the diagnosis of latent lues.
5. Discuss the treatment of high blood pressure in a woman seventy years old.
6. Discuss the etiology, symptoms and treatment of seasickness.
7. Discuss the clinical and laboratory diagnosis of leprosy.
8. Discuss eosinophilia, its significance and the method by which its presence is determined.

9. Discuss the diagnosis and medical treatment of cholelithiasis.
10. Discuss the symptoms, signs and treatment of hyperthyroidism.
11. Discuss ascites, its etiology and symptoms.
12. Discuss the etiology of diarrhoea in children.

(Answer ten questions only.)

Chemistry and Toxicology.

H. CLIFFORD LOOS, M. D.

June 12, 1917.

1. What is the general composition of Hydrocarbons of the Parafin series?
2. What is the formula of Methyl-Ethyl Ether and give general chemical characteristics.
3. What is the difference between vegetable and animal life from a chemical standpoint?
4. What is the final result of decomposition of dead plants or animals?
5. What is the treatment for the results of an overdose of Digitalis?
6. Give method of the action of Phenol introduced into the stomach.
7. Give in detail a recognized treatment for chronic Morphine poisoning.
8. Give treatment for Wood Alcohol poisoning.
9. Give a full description of the nature and action of enzymes. Give examples of each class.
10. Explain the term calorie, and give its use in the valuation of food.
11. Discuss alkaloids, name five and give their toxicology.
12. What substances poison by way of the respiratory tract?

(Answer ten questions only.)

Obstetrics and Gynecology.

For Physicians and Surgeons.

By ROBERT A. CAMPBELL.

June 12, 1917.

1. What measures should be used to prevent, (a) Mastitis; (b) Ophthalmia Neonatorum
2. Describe the operation for complete laceration of the perineum one year after injury.
3. Discuss uremic poisoning in the pregnant woman.
4. (a) What should be done prior to the application of forceps?
   (b) Describe their applications.
   (c) Describe the method of delivery after application.
5. Give technique of operation for cancer of the cervix.
6. Discuss the phenomena of menstruation.
7. (a) Under what conditions is amputation of the cervix indicated?
   (b) Why is it preferred to other methods?
   (c) Describe the operation.
8. Discuss Pelvic Cellulitis and Pelvic Peritonitis.
9. Give causes and treatment of prolapsed cord.
10. Describe the modus operandi in arm presentation.
11. Give etiology and treatment of sub-involution of the uterus.
12. What procedures may be used for the resuscitation of the child?

(Answer ten questions only.)

Anatomy and Histology.

WM. R. MOLONY, M. D.

June 13, 1917, 9:00 A. M. to 11:30 A. M.

1. Discuss cartilage, giving brief description of each of the three varieties.
2. Name structures derived from
   (a) The Ectoderm;
   (b) The Mesoderm;
   (c) The Entoderm.
3. Discuss adipose tissue.
4. Describe the sacrum.
5. Describe the maxilla.
6. Discuss a bursa. Locate ten.
7. Give path of sensory impulse from skin to brain.
8. Give path of motor impulse from brain to muscle.
9. Discuss the colon.
10. Give the gross and topographical anatomy of the pancreas.
11. In an amputation of the thigh through its middle what structures would be severed?
12. In a total amputation of the shoulder (removal of the upper extremity with the scapula from the body), what muscles would be severed?

(Be brief and to the point and arrange your answers in columns where possible.)

(Answer ten questions only.)

Homeopathic Materia Medica, Therapeutics, Pharmacology and Prescription Writing.

ROBERT A. CAMPBELL.

June 13, 1917, 1:30 P. M. to 3:30 P. M.

1. Name four remedies useful in Pneumonia with the indications for each.
2. Write a prescription for an injection for acute Gonorrhoea, with directions for use.
3. Discuss Bryonia Alba.
4. Describe the physiological action of Merc. Corr. on the intestinal tract.
5. Give the indications for three remedies for acute cystitis.
6. Name and give dosage of two remedies which will make the urine alkaline, and two which will make it acid.
7. What is apomorphine? Give dosage indications and action.
8. Give treatment for a case of La Grippe.
9. Give indications for Cactus, Digitalis, and Glonoin in heart trouble.
10. Discuss the cause and treatment of Asthma.
11. Write a prescription for three drugs and a vehicle. Describe the condition for which you would prescribe it.
12. Discuss peripheral Neuritis, and outline treatment.

(Answer ten questions only.)

Materia Medica, Therapeutics, and Pharmacology.

A. M. SMITH, M. D.

June 13, 1917, 1:30 P. M. to 3:30 P. M.

1. Outline the treatment of a case of diabetes mellitus, of two years' duration, in a man of 50 years of age, whose present weight is 150 pounds.
2. Discuss the treatment of an acute attack of bronchial asthma.
3. Define mydriatic, diaphoretic, sedative, hypnotic, oxytocic, carminative. Give example and dosage of each.
4. Discuss the treatment of myxoedema, including the symptoms of overdose.
5. What measures would you use to prevent circulatory collapse in pneumonia? In the event of its occurrence what would you advise?
6. Name six diuretics. Dose and mode of action of each.
7. Discuss digitalis, including the official preparations and physiological action.
8. In what conditions is podophyllum used? What is the physiological action? With what other drugs should it be combined?
9. Discuss the advantage of novocaine over cocaine as a local anesthetic. Why is adrenaline given in conjunction with novocaine
10. What is chrysarobin? Discuss the therapeutic use.

11. In what forms and for what conditions is arsenic commonly used? Give dosage.
12. Discuss the action of scopolamine.

(Answer ten questions only.)

Materia Medica and Therapeutics. Eclectic.

H. V. BROWN, M. D.

June 13, 1917, 1:30 P. M. to 3:30 P. M.

1. What is Opium? How obtained? Name the important constituents, artificial, alkaloids and important official preparations. State the parts affected, and mode of operation of the drug.
2. Name the various kinds of stimulants, give examples of each, and give the specific symptomatology for Nux Vomica.
3. Name the important Nitrites, the form and manner of administration, dosage and their action upon the circulatory apparatus.
4. Differentiate Digitalis, Strophanthus and Apocynum, physiologically, and give the specific indications for each.
5. Differentiate Lobelia and Ipecacuanha as to their action upon the nervous system, gastro-intestinal and respiratory tracts.
6. Name and give dosage of the active principle of Podophyllum. Give its course through, and elimination from, the body, regardless of method of administration, and its specific symptomology.
7. Differentiate between the therapeutic action of Phytolacca, Echinacea and the Iodides.
8. For what form of intestinal parasite is each of the following drugs remedial: Santonin; Spigelia; Aspidium; Felix Mas; Pelletierine Tannate.
9. Discuss the various actions of remedies upon the kidneys, and give an example of each.
10. What is an alkaloid? Name 5. What is tincture? A fluid extract?
11. Quinine: From what obtained? Physiological action in doses to 5 grs. on the blood, nervous system, heart, and respiration? How eliminated and what is its specific action?
12. Write a prescription containing two diuretic drugs and explain the action of each.

(Answer ten questions only.)

Hygiene and Sanitation.

H. V. BROWN, M. D.

June 13, 1917, 3:30 P. M. to 6:00 P. M.

1. Discuss fully the problem of controlling venereal diseases in the army.
2. (a) Enumerate five Medical Military problems of the War relating to disease.
   (b) Discuss one fully.
3. State the maximum and minimum physical requirements for military service.
4. Upon what grounds may an applicant for military service be rejected?
5. What is embraced in naval quarantine and naval hygiene?
6. Discuss prophylaxis of infectious diseases from a military standpoint, including natural and artificial immunity.
7. Describe the essentials of personal hygiene for a soldier.
8. Discuss the etiology of asthma, and the climatic therapeutics of the same.
9. (a) How may it be determined that an individual is a typhoid carrier?
   (b) How may it be determined that an individual is a diphtheria carrier?
10. Discuss the question of house dampness in its relation to disease and give methods of prevention.
11. Discuss the relation of meat to disease.
12. Discuss three varieties of stimulants excluding drugs, which are in common use.

(Answer ten questions only.)

NOTICES.

The Fourteenth Annual Meeting of the Pacific Association of Railway Surgeons will be held in San Francisco, August 24th and 25th next.

Sacramento, California,
June 23, 1917.

To the Subscribers of the Directory:

Supplements to the Directory covering data in reference to licentiates of 1917 will be furnished on receipt of seventy-five cents (75c).

C. B. PINKHAM, M. D.,
Secretary-Treasurer Board of Medical Examiners,
State of California.

DEPARTMENT OF PHARMACY AND CHEMISTRY.

Edited by FRED I. LACKENBACH.

(Devoted to the Advancement of Pharmacy and its allied branches; to the work of the Council on Pharmacy and Chemistry of the American Medical Association, and to matters of interest bearing upon therapeutic agents offered to the medical profession. The editor will gladly supply available information on subjects coming within the scope of this Department.)

NEW AND NONOFFICIAL REMEDIES.

Since publication of New and Nonofficial Remedies, 1917, and in addition to those previously reported, the following articles have been accepted by the Council on Pharmacy and Chemistry of the American Medical Association for inclusion with "New and Nonofficial Remedies":

Kephalin-Armour.—The hemostatic phosphatid obtained from spinal cord and brain tissue of mammals. It is essentially the same as Brain Lipoid, N. N. R. For a discussion of the actions and uses see New and Nonofficial Remedies, 1917, p. 124, under "Fibrin Ferments and Thromboplastic Substances (Kephalin)." Kephalin-Armour is applied freely to bleeding or oozing surfaces in 1 to 2 per cent. suspensions in physiological sodium chlorid solution. Armour and Co., Chicago (Jour. A. M. A., June 2, 1917, p. 1625).

Thorium Nitrate.—A white substance, very soluble in water and alcohol. Soluble thorium salts resemble alum in their local astringent and irritant properties. They are not absorbed from the alimentary canal. The non-precipitant double salts of thorium are practically non-toxic, even intravenously. Thorium salts are fairly radioactive.

Thorium Sodium Citrate Solution.—Prepared by dissolving thorium nitrate, 10 gm., and sodium citrate, 15 gm., in water, neutralizing with sodium hydroxide and diluting to 100 cc. Being impervious to Roentgen rays, the solution is used to obtain cystograms of the renal pelvis and urinary bladder.

Thorium Solution for pyelography—H. W. and D., 10 per cent.—It is the same as thorium citrate solution. Prepared by Hynson, Westcott and Dunning, Baltimore, Md.

Stronger Thorium Sodium Citrate Solution.—Prepared by dissolving thorium nitrate, 15 gm., sodium citrate, 22.5 gm., in water, neutralizing with sodium hydroxide, and diluting to 100 cc. It is used for obtaining urethral pyelograms.

Thorium Solution for Pyelography—H. W. and D., 15 per cent.—It is the same as thorium citrate solution. Prepared by Hynson, Westcott and Dunning, Baltimore, Md. (Jour. A. M. A., June 16, 1917, p. 1817).

Betanaphthol Benzoate—Anthony-Hammond Chemical Works, Inc.—A brand of betanaphthol benzoate which complies with the N. N. R. standards for this drug. Anthony-Hammond Chemical Works, Inc., New York City.

Calcium Cacodylate.—The calcium salt of cacodylic acid containing from 43.5 to 48 per cent. of arsenic in the form of cacodylic acid and free from arsenite, arsenate and monomethylarsenate. It has the mild arsenic action of cacodylates. Calcium

cacodylate is white, almost odorless, and very soluble in water.

Ampuls Calcium Cacodylate Solution—Mulford. —Each ampule contains calcium cacodylate 0.045 gm. in 1 cc. The H. K. Mulford Co., Philadelphia, Pa.

Chlorazene Surgical Cream.—It contains chlorazene, 1 gm., in 100 gm. of a base composed of sodium stearate, 15 per cent., and water, 85 per cent. The Abbott Laboratories, Chicago.

Borcherdt's Malt Extract with Cod Liver Oil.— A liquid composed of cod liver oil, 20 per cent., and Borcherdt's Malt Extract Plain, 80 per cent. The Borcherdt Malt Extract Co., Chicago.

Borcherdt's Malt Extract with Creosote.—100 cc. contain beechwood creosote, 4 minims per fluidounce, in Borcherdt's Malt Extract Plain. The Borcherdt Malt Extract Co., Chicago.

Borcherdt's Malt Extract with Cascara Sagrada. —100 cc. contain cascara sagrada, 60 grains per fluidounce, in Borcherdt's Malt Extract Plain. The Borcherdt Malt Extract Co., Chicago (Jour. A. M. A., June 23, 1917, p. 1911).

Lipoiodine-Ciba.—The ethyl ester of iodobrassidic acid containing 41 per cent. of iodin. Lipoiodine-Ciba is odorless, tasteless, insoluble in water but very soluble in fatty oils. When administered, it is absorbed almost completely and excreted more slowly than inorganic iodids but more rapidly than with other iodized fats. It is said to be less likely to produce gastric irritation than ordinary iodids. It is supplied only in the form of Tablets Lipoiodine-Ciba, 0.3 gm. A. Klipstein and Company, New York (Jour. A. M. A., June 30, 1917, p. 1985).

Items of Interest.

The Calcium Content of the Blood.—It has been found that the calcium content of the blood plasma of cattle is remarkably constant, even when there is a continuous withdrawal as a result of pregnancy or lactation. It has also been found that there is no marked deviation from the normal in the calcium content of the blood serum of patients in the various stages of pulmonary tuberculosis. Even when a high milk diet was furnished over long periods, the calcium content of the blood was not increased above normal. Further, it was shown that the calcium content of the blood serum of normal human adults did not differ from that in sufferers from tuberculosis. Finally, it has been found that the calcium content of blood plasma differs little from the normal in advanced cases of uremia or in hemophilia or in purpura hemorrhagica (Jour. A. M. A., June 23, 1917, p. 1915).

Russell Emulsion and Russell Prepared Green Bone.—The Council on Pharmacy and Chemistry reports that "The Russell Emulsion" and "The Russell Prepared Green Bone," put out by the Standard Emulsion Company, are inadmissible to New and Nonofficial Remedies. The Russell Emulsion is said to be composed of beef-fat, coconut, peanut and cottonseed oils, held in suspension by albumin. The mixture is called a "physiological" emulsion and is exploited on the theory that lime starvation is a main factor in tuberculosis and that large amounts of fat are required for the lime-starved. There is no proof that tuberculosis is due to an insufficiency of lime in the tissues, and the claims made for the emulsion are grossly unwarranted. Particular attention is called to the exploitation of the emulsion by one Dr. Hague who talks before medical societies. The Russell Prepared Green Bone is said to be made by digesting chicken bones with hydrochloric acid and pepsin and adding glycerin at the end of the digestion. This is advertised as a lime food. The greater value of a few glasses of milk daily is not mentioned (Jour. A. M. A., June 23, 1917, p. 1931).

NEW MEMBERS.

Allen M. Thomson, Sonoma.
Herbert I. Bloch, San Francisco.
Charles A. Bell, Santa Barbara.
Laurence E. Heiges, Lompoc.
Henry Clay Bagby, Santa Barbara.
Augustus Lincoln Morrill, Antioch.
Bartholomew Gattuccio, Davenport.
Bessie Faith C. M. Raiche, Balboa.
Oscar O. Young, Garden Grove.
Albert Osborne, Anaheim.
George H. Dobson, Santa Ana.
Fred. O. Butler, Eldridge.
Charles R. Knox, El Cajon.
Chas. Lincoln Stoddard, San Diego.
Carrie H. Edwards, San Diego.
H. Clifford Loos, San Diego.
Arthur Newton Bobbitt, San Diego.
Frank Dunlap, Brawley.
George Wirt Hathaway, Yreka.
Wilfred E. Bixby, Sebastopol.
Howard Hill Markel, San Francisco.
William H. Harrison, San Francisco.

DEATHS.

CHARLES A. VON HOFFMAN, M. D.

In Memoriam.

The death of Dr. Charles von Hoffman on May 11th closed a long and honorable career; and for those who knew him it carried a special poignancy. Dr. von Hoffman was graduated from the University of Leipzig in 1875; he established himself in San Francisco in the following year. His temperament and talents were eminently suited for the branches which he was to make his life work, and within a few years his reputation as a gynecologist and obstetrician of the first rank was firmly established. In 1890 he joined the faculty of the medical department of the State University, and later succeeded the late Beverly Cole as head of the department of gynecology. Five years ago he became emeritus professor. He was also closely identified for many years with the Alexander Maternity of the Children's Hospital and other institutions.

In an age of riotous enthusiasms Dr. von Hoffman brought to bear on his work and conduct cautious and critical discrimination. In this sense alone was he a conservative. His lectures epitomized shrewd first-hand clinical observations enriched by the fruits of wide reading, and his surgical work was characterized by a uniformity of technical excellence and good judgment. Those who had the privilege of his friendship realized the extent and variety of his knowledge, his luminous and delicate insights, and what was perhaps more remarkable than any single faculty, the admirable harmony and symmetry of his mind and character. And we cannot forget those higher attributes which made him universally respected—his fervent love of truth, his wide tolerance and his disdain for noisy triumphs. In his walk through life there was no obtrusiveness, no elbowing, none of the artifices which bring forward little men.

What remains of one in memory, after death, is usually but a slight thing—the phantom of an attitude, the echo of a phrase, or some victory in a critical hour. It is as if the whole meaning of a man's life had now shrunk to a mere gesture, act or word suggestive of his singularity. Fortunate are those whose personality and achievements have been sufficiently pronounced to be victorious over the pity of such a diminution and abridgment. For many of us such a man was Charles von Hoffman.

A. J. L.

Edwin Dunbar Farrow, Visalia.
John Montgomery, San Francisco.
Russell D. Adams, Monrovia.
James Watson Wood, Long Beach.
C. Guy Reily, Los Angeles.
Harriet F. Pillsbury, Berkeley.